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SPONTANEOUS PNEUMOTHORAX IN ASIAN INFLUENZA

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The epidemic of Asian influenza was at its height in the Transvaal Province, South Africa, between July and August 1957. Two hundred nurses were admitted to the General Hospital, Pretoria, suffering from this condition. Two of them developed a spontaneous pneumothorax and are the cases reported below.

CASE REPORTS

Case 1

E.W.O., aged 17, was admitted to the Pretoria General Hospital on 17 August 1957 as a case of Asian influenza. She had complained of malaise, headache, feverishness and muscle pains. Her temperature was 100°F, pulse rate 104 per minute, and respiration rate 24 per minute. She was given Sulphatriad and penicillin.

On 18 August crepitations were heard at the bases of both lower lobes; temperature 101°F, pulse rate 128 per minute, respirations 32 per minute. Her temperature rose to 103°F that evening. Next day, widespread rhonchi were heard on auscultation of the lungs.

On 20 August she became critically ill. She was orthopnoeic, cyanotic, cold and clammy and surgical emphysema was present anteriorly in the neck. The sputum was frothy and stained pink. A portable X-ray plate (Fig. 1) showed bilateral patchy consolidation, atelectasis of the right lower lobe, and a pneumothorax on the same side. The presence of surgical emphysema was confirmed.

Her critical condition remained unchanged. Continuous oxygen was administered. In view of the large pneumothorax and her respiratory distress a catheter was introduced into the pleural cavity *via* the 2nd right intercostal space anteriorly and connected to an under-water drainage system. The pressure in the pleural cavity was positive. There was a slight improvement in her breathing after release of the tension; this improvement, however, was not maintained and she died on 20 August some 4 hours later.

Case 2

A.M.C.W., aged 23, was admitted to the hospital on 21 August 1957. One week before admission she had had influenza but had not taken it seriously. For 3 days before admission she had had a retrosternal pain and had developed a troublesome cough.

On examination she was obviously ill-looking and there was a tinge of cyanosis in her lips and finger-tips. The pulse rate was 120 per minute, respirations 28 per minute and the temperature 101°F. Crepitations were heard at both lung bases.

Aureomycin and a stimulant cough mixture were given.

By 23 August her general attitude was one of anxiety and fear, with a complaint of intense pleural pain. Cyanosis was more obvious and orthopnoea more pronounced, the respirations being 36-40 per minute. The temperature was 103°F and the pulse rate 140 per minute. Examination of the chest revealed dullness to percussion over both lung fields posteriorly, and on auscultation bronchial breathing was heard in these areas. A portable X-ray plate showed bilateral bronchopneumonia. Albamycin, penicillin and ACTH were given.

Next day she began to cough small quantities of thick, blood-stained, purulent sputum. Her temperature remained high over the next 3 days and pleural pain was intense. A pleural rub was detected on the left side.

On 27 August her temperature fell to 98°F, her pain lessened and her appetite returned. Sputum culture grew a coagulase-positive staphylococcus sensitive to chloromycetin, streptomycin, albamycin, sulphonamide, erythromycin and polymyxin. Albamycin and aureomycin were replaced by chloromycetin and

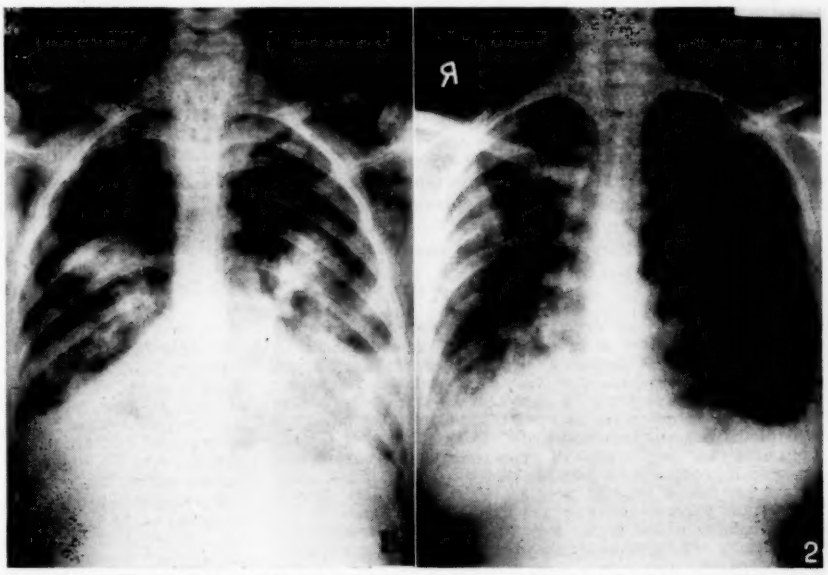

Fig. 1. Case 1. Bilateral patchy consolidation, atelectasis of the right lower lobe and right-sided pneumothorax. Surgical emphysema is present. *Fig.* 2. Case 2. Left-sided tension pneumothorax. There is an effusion visible in the left costo-phrenic sulcus. The pneumatoceles in the compressed left lung can be seen.

streptomycin. An X-ray plate showed numerous pneumatoceles in both lungs varying in size from 1 to 5 cm.

On 30 August a dramatic deterioration took place in her condition. She became even more short of breath, cyanosed again, cold, clammy, and extremely frightened. There was hyper-resonance over the left side of her chest with displacement of the trachea to the right. A portable X-ray plate confirmed the presence of a left-sided tension pneumothorax (Fig. 2). A catheter was introduced into the left pleural cavity *via* the 2nd intercostal space anteriorly and the tension was relieved by drainage of the air under water. The patient improved dramatically and a follow-up X-ray plate showed that the lung had re-expanded. The improvement, however, was short-lived, and she passed into a state of peripheral circulatory failure, dying on the following day (1 September).

DISCUSSION

Air finds its way into the perivascular tissue planes of the lung *via* ruptured alveoli, bronchioles or a bronchus and spreads towards the mediastinum. Its path may then follow the fascial plane of the great vessels into the neck and present clinically as surgical emphysema. A build-up of pressure in the mediastinum may, however, result in a rupture of the pleural covering with the development of pneumothorax.[1]

It is highly likely that this was the sequence of events in case 1. Air rarely leaks into the interstitial pulmonary tissues without predisposing causes. In this instance atelectasis of the right lower lobe, with over-inflation of adjoining areas, bronchospasm and cough, are sufficient factors to predispose the lung to this condition. A pneumatocele bursting into the interstitial lung tissue would have been a ready explanation for the sequence of events in this case. The radiological decision whether pneumatoceles had formed in the right lung was made difficult by the presence of surgical emphysema.

The spontaneous pneumothorax in case 2 was the result of rupture of a pneumatocele into the left pleural cavity. Pneumatoceles forming in areas of consolidation, and resultant spontaneous pneumothorax from their rupture, are characteristic radiographic findings of staphylococcal pneumonia.[2] Staphylococcal pneumonia is rare but seems to occur most commonly as a complication of influenza.[3]

In the 1957 epidemic of Asian influenza *Staphylococcus aureus* was the most dangerous secondary invader.[4,5] The sputum culture of a coagulase-positive staphylococcus and characteristic radiographic signs of pneumatoceles in this patient confirmed the diagnosis of secondary invasion. That the improvement after release of the tension within the pleural cavity was only temporary indicates the overwhelming nature of the combined infection.

Cases of influenza secondarily infected by the staphylococcus may die at the stage of acute tracheo-bronchitis.[5] Bronchopneumonic changes, however, may herald a train of events as here described and will help to indicate those cases of Asian influenza which are not following the expected benign course.

SUMMARY

1. Two cases of Asian influenza complicated by spontaneous pneumothorax are described.

2. The probable mechanism of the development of spontaneous pneumothorax in each case is discussed.

3. The danger in Asian influenza of the secondary invasion by coagulase-positive staphylococci is emphasized. Such invasion may alter the course of a relatively benign illness into a potentially fatal one.

OPSOMMING

1. Twee gevalle van Asiatiese griep, gekompliseer deur spontane pneumotoraks, word beskryf.

2. Die waarskynlike meganisme van die ontwikkeling van die spontane pneumotoraks in albei gevalle word bespreek.

3. Die gevaar by Asiatiese griep van die sekondêre inval deur koagulase positiewe Stafilokokke word beklemtoon. So 'n inval mag die verloop van 'n betreklik onskadelike siekte tot 'n potensiële dodelike een verander.

I should like to thank Dr. J. D. Verster, Deputy Superintendent of the Pretoria Hospital, under whose care these patients originally fell, for permission to publish, Dr. J. Hough for the use of his clinical notes in case 2, and Mr. Theo Marais, head of the Photographic Department of the University of Pretoria, for the photographs of the X-ray plates. Dr. J. C. van der Spuy, head of the Department of Thoracic Surgery, has greatly encouraged the writing of this paper and for his help I should like to express my special thanks.

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THE FINGER-PRINT SWEAT TEST : A MODIFIED METHOD

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In a previous paper[1] we described the Shwachman finger-print test for sweat chlorides as a specific screening and diagnostic test in cystic fibrosis of the pancreas. A finger impression is taken on an agar plate in which silver nitrate and potassium chromate are suspended. Varying degrees of bleaching occur on the surface of the plate, depending on the concentration of chloride in the sweat of the finger. This is recorded as 1+, 2+ or 3+. We emphasized that only a case of cystic fibrosis of the pancreas is capable of producing 3+ bleaching *within* 20 *minutes of washing of the hands free of chloride*, with a few exceptions described in the previous paper. However the agar medium tends to dry out so that the record is not a permanent one. To overcome this difficulty we have suspended the ingredients in chloride-free filter-paper.

Whatman No. 1, 9-cm. filter papers are dipped into 1% solution of silver nitrate and dried in an oven at 110°C in the dark for 30 minutes. They are then dipped into a beaker containing 2·5% solution of potassium chromate for ½ minute. This dipping must be done in one quick movement to prevent streaking. The papers are then quickly washed in distilled water, followed by 96% ethanol, and finally dried in the oven at 110°C for 15 minutes. If exposed to the light the papers tend to darken, but this does not apparently influence the reaction with Cl ions.

Before use, one drop of either chloride-free buffer or distilled water is placed on the impregnated paper and allowed to spread evenly. A finger is then pressed *gently* onto the damp surface. As soon as the paper is dry the degree of bleaching is read exactly as in the original agar method. *It must be emphasized that in the presence of a* 3+ *reaction, the patient's hands must be thoroughly washed and the test repeated* 20 *minutes later.*

We have checked the method against the original agar method described by Shwachman and the results appear to be identical. The advantage of the filter-paper modification is that the recording appears permanent and can be filed away with the patient's clinical record. Notes may be written on the filter paper with a ball-point pen.

Thanks are due to Prof. J. G. A. Davel for access to his cases and encouragement with the project; and to Dr. T. Gerritson of the National Nutrition Research Institute, C.S.I.R., Pretoria, for his technical assistance in preparing the filter papers.

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EDITORIAL

DIABETIC EMBRYOPATHY

The diabetic mother frequently gives rise to a foetus with several pecularities. A high proportion of her infants are overweight—they often exceed 10 lb.— and this increase in weight is made up of several components. The babies are both over-long and over-fat; they are oedematous and characteristically lose a pound or so of fluid weight during the first few days of life. Their internal organs are large, in particular the heart and liver. They may have an expanded erythron, with widespread haematopoiesis and a high haemoglobin content in their blood. Their general appearance of fat, flabby, rubicund weakness may closely resemble the picture of Cushing's syndrome.

These babies, and also those which escape being overlarge, behave as if they were feeble, undersized, premature infants; they have difficulty in breathing and sucking, and easily regurgitate fluids into their lungs. They are susceptible to birth trauma, infection and hyaline membrane disease so that they may die within the first few days of life unless specially protected. Although their blood-sugar levels may be very low soon after birth, this is probably of no significance, since a normal baby may likewise have extreme hypoglycaemia without any symptoms from it.

Foetuses of diabetic mothers show a greatly increased incidence of major congenital anomalies which, like anencephaly and ectopia cordis, may be incompatible with life. Apart from such anomalies, of course, a number of foetuses are not born alive. They die around the 36th week or earlier and are delivered in a macerated state, or they die nearer term and are obtained in a better state of preservation. Although these stillbirths are near term or even at full term, they are distinguished by active hepatic haematopoiesis, similar to that seen in premature and erythroblastotic infants. Luteal cysts may be found in the ovaries. Warren and Le Compte,[1] and Cardell,[2] in particular, have described the pathological features of these infants in more detail. Probably the most interesting abnormalities are those of the pancreas. Almost invariably the islets of Langerhans in these pancreases are large, and occupy an excessive part of the whole organ. There is both cellular hyperplasia and hypertrophy. Further than this, there is an increased proportion of beta cells in the islets. In the normal newborn infant the alpha cells comprise 60-70% of islet tissue; the ratio of alpha to beta cells is reversed in the infant of the diabetic mother. The histological sections may, moreover, appear to show an unusual degree of granularity in the beta cells, which may indicate an excess of insulin content. In fact, if we take all these items together it looks as if the pancreas of the diabetic's infant may contain up to 30 times the normal amount of insulin.

This abnormal condition of the embryo, or 'embryopathy', as it has rather unfortunately been called, owes its existence to some abnormality in the diabetic mother, since it is not seen in the infants of diabetic fathers. Certainly it has been found that the birth weights of the infants of diabetic fathers are larger on the whole than those of non-diabetics,[3] yet this increase in birth weight is not nearly as striking as is found in the infants of diabetic mothers, and there is no increase whatever in the stillbirth rate in the progeny of diabetic fathers.

Now what types of diabetic are liable to such an embryopathy as has been described? Here we come across some inexplicable discrepancies. In most centres of the world the same type of baby has been found to be produced by the severe, insulin-requiring, growth-onset diabetic, by the mild, diet-controlled diabetic, and even by the prediabetic several years before she shows any obvious metabolic carbohydrate disorder. In Boston and Brussels, apparently, the tendency for foetal loss to occur in the mild diabetic and in the prediabetic is very much less, and the data from these cities seem to be unimpeachable. In Cape Town evidence was found that the tendency to produce stillbirths and large babies extended back indefinitely into the past obstetric history of the diabetic woman, while in her prediabetic phase.[3]

In fact all the above-described features of the diabetic's infant apply with equal force to the infant of the prediabetic. It was van Beek[4] who first pointed out that the enlarged islets of Langerhans were to be found in the stillbirths of women who only later became diabetic. Woolf and Jackson have confirmed this.[5] The mean percentage of islet tissue in stillborn pancreases of control infants was found to be 1·3% by their method, while it was around 7% in erythroblastotics, in children of diabetic mothers and in children of prediabetic mothers. It is extraordinary that once again this similarity is seen between erythroblastosis and diabetes. No other cause for such enlarged islets in the stillborn infant is known. In fact, the finding of enlarged islets in a stillborn which is not erythroblastotic and whose mother is not an overt diabetic is the best possible indication of prediabetes in the mother. Woolf and Jackson's results illustrate this: Pancreases from 109 autopsies on stillbirths were specially examined and their islet contents estimated; 18 were found in which the proportion of islet tissue was over 5·5%, apart from those cases which were known to suffer from Rh incompatibility or whose mothers were diabetic at the time of pregnancy. Of the 18 relevant mothers 12 were traced for follow-up; 5 were found to have become diabetic and 5 gave slightly abnormal glucose curves; in the other 2 there was strong collateral genetic and obstetrical evidence of prediabetes. Thus all 12 mothers were probably prediabetic when they gave rise to the stillborn infants with large islets of Langerhans.

Looked at from another angle, these findings may provide a pointer in hitherto unexplained stillbirths, since the finding of large islets will indicate that maternal diabetes or prediabetes has played a part in the foetal death.

These excursions into the realms of prediabetes may help us in our search for the cause of this strange embryopathy.

It is certainly most unlikely that maternal hyperglycaemia can play any part, since many prediabetics show normal blood-sugar levels during pregnancy, and yet produce abnormal infants. A sensitivity to or excessive production of growth hormone or of glucocorticoids in the diabetic pregnancy has been suggested, and, although Professor Hoet[6] has made some interesting experiments the results of which favour the latter, the evidence is as yet not very good in favour of either. Although the plasma-cortisol level rises during all pregnancies, the levels observed are no higher in the diabetics. There is no tendency for women who are in the early stages of acromegaly or Cushing's syndrome to produce large babies or stillbirths,[7] and this is surely strong evidence against the view that either growth hormone or corticoids are the sole cause of the embryopathy. The large pancreatic islets suggest an excessive stimulation of this tissue. Is the infant's own insulin, acting as a 'growth hormone' itself, in fact the stimulus to the excessive size? And what is the connexion with the large islets in erythroblastosis?

Turning finally to the prevention and management of the embryopathy, in some centres insulin is being given to the prediabetic during pregnancy, even when her blood sugar is normal, in the hope that it may be effective in preventing some of the features of the embryopathy. And yet in the established diabetic, the very best possible control of the mother's diabetes will reduce the incidence of abnormal babies only partially. To obtain a live child it appears to be even more important to induce labour early if the foetus is judged to be large enough, and to manage the baby exactly as though it were truly a premature infant. Before this can be done, of course, it is necessary to make a diagnosis of maternal diabetes or prediabetes, and it is likely that a number of babies may be saved if the latter diagnosis is suspected and the suspicion is acted upon.

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VAN DIE REDAKSIE

DIE SWAAI VAN DIE PENDULUM IN DIE VERLOSKUNDE

Ons lees gedurig van die swaaiende pendulum in die geskiedenis, maar in geen ander vertakking van die medisyne is dit so opmerklik soos in die verloskunde nie. In die twintiger- en dertiger-jare was die verloskunde daarop toegespits om die moederlike sterftesyfer te verminder terwyl die kind van ondergeskikte belang was. Vandag egter, waar die moederlike sterftesyfer gedaal het tot 'n minimum, is alles weer daarop toegespits om die foetale sterftesyfer af te bring tot 'n minimum. Twintig jaar gelede was dit byna krimineel om 'n keisersnee to maak, en alles is gedoen om 'n vaginale verlossing te bewerkstellig. Vandag word 'n keisersnee baie makliker aangedurf.

Die vraag wat ons dus in die gesig staar is of dit nie tyd is vir die pendulum om terug te swaai nie? Of het dit alreeds te ver geswaai? Word daar nie misbruik gemaak van keisersneë nie? Met die moderne antibiotiese middels, die verbeterde hospitaalgeriewe en die gemak waarmee 'n keisersnee uitgevoer kan word, word daar vandag dikwels nie twee maal gedink voordat 'n keisersnee uitgevoer word nie. Is die tyd van die bekende geduld van die verloskundige dan verby? Gaan die moderne verloskundige dan die kuns van die verloskunde verloor? Die ou gesegde dat ‚die bekwaamheid van 'n verloskundige beoordeel word volgens die manier waarop hy 'n stuit verlos' bestaan amper nie meer nie want hoe dikwels word 'n keisersnee nie gedoen met net 'n stuitligging as indikasie nie?

In hierdie eeu van meganisasie en outomatisasie moet ons egter nie vergeet dat die natuur altyd die beste is nie. Indien 'n vaginale verlossing moontlik is, is dit nog, ten spyte van al die bykomstige tegniese hulpmiddels, vir sowel die moeder as die baba die beste. Die moederlike en foetale mortaliteit by keisersnee is onder die beste omstandighede nog steeds baie hoër as by vaginale verlossings. Ons noem net 3 voorbeelde waaraan die keisersnee die moeder en die baba meer blootstel, naamlik: ruptuur van die uterus met daaropvolgende swangerskappe, pulmonêre embolisme en hyalien-membraan.

Wanneer 'n keisersnee gedoen word vir die jong vrou wat nog 'n lang vrugbare tyd voor haar het, vergeet ons dikwels hoe algemeen ruptuur van die uterus in die daaropvolgende swangerskappe is. Syfers in die literatuur variëer vanaf 0·25%[1] tot 3·3%[2] vir die laer segment, en 2·2%[3] tot 4·2%[4] vir die klassieke keisersnee. As ons dan verder daarop let dat die moederlike mortaliteit by ruptuur van die uterus vanaf 4% tot 50%[4] varieër en die foetale mortaliteit vanaf 40% tot 90%,[4] dan laat dit ons bepaald met angs terugkyk na ons maklike besluite in die verlede. Is ons dan geregverdig om so maklik oor te gaan tot 'n keisersnee as ons die moeder en die baba in daaropvolgende swangerskappe aan so 'n groot gevaar blootstel? Dit is wel waar dat hierdie syfers onder moderne hospitaalbehandeling verminder kan word, maar hoeveel van die verloskunde in ons land word nie nog in huise en onder baie primitiewe toestande gedoen nie?

Die voorkoms van pulmonêre embolisme na normale geboortes variëer vanaf 1 in 3,000[5] tot 1 in 10,000,[6] terwyl dit na buikoperasies — keisersnee ingesluit — variëer vanaf 1 in 400[5] tot 1 in 550. Hier weer, dus, is die kanse van die pasiënt wat 'n keisersnee moet ondergaan 6-20 keer groter as by spontane geboorte om hierdie gevreesde komplikasie te kry.

Wat van die foetus? Dit is keer op keer bewys dat selfs vir die te vroeggebore baba 'n vaginale verlossing beter as 'n keisersnee is. Dit is bevind dat 30%[7] van neonatale sterfgevalle die gevolg van hyalien-membraan is. Hierdie toestand kom grotendeels voor by babas wat met keisersnee gebore is en sommige skrywers beweer selfs dat dit *net* by sulke gevalle voorkom. Moet ons dan nie vra of ons dit nie aan onsself te wyte het dat die bogenoemde toestand vandag so algemeen geword het omdat ons so baie keisersneë doen nie?

Ons wil nie aanbeveel dat ons na die tydperk van 30

jaar gelede, toe alle soorte gevreesde vaginale manipulasies uitgevoer is om vaginale verlossings te bewerkstellig, moet teruggaan nie. Ons moet egter 'n meer gematigde houding in hierdie opsig inneem. In ons land met sy wyduitgestrekte vlaktes waar die verloskunde nog so dikwels onder baie primitiewe toestande moet geskied, moet ons bepaald nie die behendige hande van die knap verloskundige deur onbruik verlore laat gaan nie.

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CEREBRAL VASCULAR DISEASE: THE SURGEON'S INTEREST*

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Although I am speaking as a surgeon dealing with cerebral vascular disease, it would be truer to say that the cerebral vascular system deals with the neuro-surgeon. In general we can achieve technically only as much as this system will allow us. Tumours, inflammations and injuries all declare themselves by primary or ultimate interference with cerebral circulation, and our manipulative intervention depends for success upon the extent to which we can correct this circulatory distress. In this short discourse I am concerned, therefore, with cerebral vascular disease in the widest sense; that is, whether the circulation is upset directly by blood-vessel pathology, or secondarily by other disease within the cranium.

There are grave difficulties confronting us in our appreciation of the remote and complex vascular effects of any disease process, and even greater difficulties in judging the need for interference, or the best time for intervention, and also in the choice of methods that will accord with natural circulatory requirements.

Since Cushing's time, the advances in neuro-surgery have mainly depended upon better understanding of the dynamic interrelationships between every component in the cranium. Our approach, however, is still in the main far too anatomical and static. As an investigation angiography has helped us by its simultaneous indication of the site and extent of gross disease, but it fails to reveal the more subtle defects of circulation that are allied to the functional rather than to the structural physiology. Its most serious limitation as a test is that it demonstrates a state of the moment and is therefore unrevealing of the more subtle qualities of reserve and adaptability of the brain as a whole under varying stress conditions.

A further fundamental difficulty in intracranial surgery is that what we assume to be a state of balance within the closed cranium has to be reviewed under circumstances of open operation, where the pressure equilibrium of the blood and cerebrospinal-fluid systems are immediately altered providing a vent to the atmospheric exterior, or the alteration of intervening tissue resistances.

CEREBRAL ANEURYSM

I will take as an example of the effects of this change of habitat the cerebral aneurysmal sac. Most surgeons have, more often than they care to remember, experienced the spontaneous rupture of a sac when it has been stripped naked of its surrounding tissue and fluid support, even before the sac itself has been subjected to handling. A not dissimilar example is the more frequent occurrence of peri-arterial haematoma after carotid arterial puncture in cut-down techniques of angiography as opposed to the percutaneous methods, where the integrity of the surrounding tissues and muscle tone have been maintained.

These experiences have made one feel, especially with aneurysm, that the extra-arterial pressure environment is of paramount importance. In the intact cranium this environment could be expected to be a natural medium for the control of a pressure gradient across the sac wall, so as to prevent its thinning and rupture. This indeed must happen, considering the surprisingly long life of a congenital sac before it ruptures. In the same way as an adverse pressure gradient can be rapidly induced at operation, so in a natural way it can arise within the intact cranium as a result of more insidious aging or of disease which produces softening of tissues by demyelination or the lessening of turgidity of these tissues by dehydration or other means. It is probable that we have under-estimated the importance of those changes which predispose a sac to sudden thinning and rupture upon the application of a minor transient rise of the intrinsic blood pressure.

On the other hand, immediately after rupture the external pressures tend to rise by the formation of surrounding blood clot, cerebral oedema, venous hypertension, and raised pressure of the cerebrospinal fluid. This readjustment by nature serves in many instances to check the extravasation of arterial blood, even while the hiatus in the sac remains potentially patent; otherwise bleeding would be more destructive than it usually is. Were these natural defences to continue long enough, the weak spot in the sac would have time to heal; but should they melt away prematurely, the reversal of the pressure gradient would allow a subsequent bleed to occur.

If these arguments are valid we could devise a more rational perspective of approach to the management of aneurysms. Their application would be more helpful than statistical guides in determining the prognosis of a given case. For instance, it would be better to sustain these natural defences and initially withhold surgical intervention after a first bleed. For the first few days at least we should try to maintain in the closed cranium for as long as possible a reasonable state of hypertension above that of the estimated intrasacular tension (i.e., the normal blood pressure). The longer we can maintain this balance the more likely is the sac to reach a safe stage of healing. If we find on lumbar puncture that the intracranial pressures are dropping too rapidly to normal or sub-normal, we should realize that this is a case very likely to bleed again before the healing of the sac has progressed sufficiently. In this interval, then,

* A paper read in plenary session at the South African Medical Congress, Durban, September 1957.

we should declare for a more direct attack upon the sac without reference to arbitrary timing.

Surgical Practice

It would be fair to review practical current opinion at this point. Provided the sac is accessible in position and capable of isolation and the general intracranial conditions will allow a safe approach, environmental considerations can be ignored in favour of immediate direct surgery.[1] Usually, however, the state of the brain is such that for many days early surgery creates a morbidity as great as the mortality caused by waiting. Norlén and Olivecrona[2] would therefore advocate conservatism even into the 2nd and 3rd week.

Our writings and thoughts up to now have been preoccupied with the position of the sac from the point of view of its surgical accessibility, the intrinsic pathology of its wall, and its internal pressures. Perhaps we have not put sufficient emphasis on the extrinsic conditions influencing the environment, and the possibilities of developing an intelligent management of these external factors. Until we have learnt more about the assessment and management of the environment of the aneurysm along these lines, the ideal treatment remains the direct isolation or obliteration of the sac or, failing that, the reinforcement of its wall from outside by the use of muscle, different fibre materials, acrylic resin, and so on. Unfortunately, the inaccessibility of the aneurysm sometimes makes the direct attack impossible. Certainly this is so in many aneurysms in the early stages up to about a week after a 'massive bleed', when high intracranial pressure, cerebral oedema, and the obscuring of cleavage lines by blood pigment persist. The anterior carotid aneurysm in particular, while reasonably accessible, is so often fusiform that isolation is not possible without risking the obliteration of its parent anterior cerebral vessels, and this in particular remains our 'problem child'. Here we often have to resort to the reinforcement of the wall by the materials mentioned above, and in large measure one feels that this manoeuvre is not so satisfactory as we are inclined to think at the time we carry it out. In the majority of instances there is no very good evidence that the sac is in fact reinforced; muscle does not seem to graft naturally upon the exterior of an artery and I do know that quite a few of our cases have subsequently bled fatally from the same site.

It is not feasible to reduce the intrinsic blood pressure of the sac for any length of time. We may do this temporarily by using a hypotensive technique at the time of operation. But this supportive method is, to my mind, fraught with very real dangers to the general cerebral circulation, the complications of which are coming more and more to our notice. Hypothermia is a much more physiological adjunct in this regard. Attempts to reduce the blood pressure for a longer period of time by carotid ligation are also in the main unsatisfactory substitutes for the direct attack. Supposing that the systolic pressure could thereby be reduced to an effective level for a period long enough for the sac to heal, it could not be expected to maintain an adequately low pressure with safety for an indefinite period, and two questions in this regard remain unanswered:

1. Is an adequately low pressure in fact produced and maintained for any sufficiency of time?

2. If it were in fact so provided, would that not mean in itself that there is an inadequate collateral supply to a lesser or larger portion of the brain, so that, even if no symptoms of distress appear initially, the patient's reserve in the face of subsequent aging or disease or stress may be seriously prejudiced?

COLLATERAL CIRCULATION IN THE BRAIN

Having hinted at the global aspects attending this focal vessel disease I pass on to consider another total vascular mechanism, the understanding of which allows us to manage problems involving occlusion of major cerebral arteries.

It has been noted repeatedly that after deliberate or spontaneous occlusion of a major artery, such as the middle cerebral or internal carotid artery, there are often surprisingly few symptoms of brain disturbance in proportion to the distribution of the vessel concerned—sometimes no symptoms at all. It is by no means inevitable that an infarct of any degree should occur in any part of the brain to which the affected vessel is assigned. If it were that the collateral supply depended upon the subsequent opening up of new channels, either to link the proximal to the distal branches of the blocked artery, or from neighbouring arteries to the distal branches, a time lag would occur which would certainly lead to recognizable neuronal death. On the contrary, compensation is usually so rapid and complete as to suggest that the requisite channels are patent and available to the blood flow at the moment of the insult to the major vessel; all that is needed to ensure the maintenance of proper metabolism is to alter the direction of blood flow and impel it with sufficient linear velocity. It might be said that 'we live normally upon our cerebral collateral circulation'; so that we have the almost maximal reserve immediately available in the event of an insult.

A large-scale example of this principle is provided under normal conditions by the existence of a separate flow within the vertebral and carotid systems, although these are linked together by a widely open posterior communicating artery and the circle of Willis.[3] The separate streams do not mix and the blood in the posterior communicating artery has no linear velocity. Any greater call upon the circulation of one or other of these systems is followed by an immediate directional change of flow through the posterior communicating artery, maintained with the full force of the blood pressure. Beever,[4] in 1909, made the postulate that collateral supply depended less upon opening up of branches of an occluded artery than upon the opening of links from adjacent vessels, and recent angiography studies support this contention by showing, in occlusion of the middle cerebral artery, the filling of the distal branch from adjacent collaterals distal to the block.

Use of this mechanism, for the most part unwitting, has stood the surgeon in good stead where he has been forced, for one reason or another, to occlude a major vessel, and has enabled him to operate more physiologically, yet in wider scope. The principle applies in varying degree in the relationship between the vascularity of a cerebral tumour and that of its matrix, survival or death of portions of the tumour being influenced by the effect of surgery or radiation therapy upon the collateral circulation remaining to the tumour. In a reverse sense the principle also applies to arteriovenous malformation where, by virtue of large open redundant collateral channels, blood-robbing takes place from the normal arterial fields, and where our clue to the surgery of these defects would be to interrupt these redundant links (providing

they can be identified and isolated) rather than attempt wholesale occlusion of the normal feeding parent vessels.

In general, at the time of the insult to major vessels, conditions may exist which prevent the collateral supply from remaining effective. Compression of the major or minor collateral channels by space-occupying lesions, such as intracerebral clots, cerebral oedema, or excessive intracranial hypertension, may have the effect of delaying or limiting adequate collateral blood flow; and here the surgeon has an active role to play to offset these adverse conditions as soon as possible—a role which extends into more medical conditions such as the encephalitides. Sometimes a very dramatic and sudden improvement occurs in a hemiparesis or an aphasia, for instance, after the reduction of intracranial pressures by single or repeated lumbar punctures or ventricular tappings, an improvement on occasions so immediate that vasospasm is unlikely to be the sole cause of the deficiency. Vasospasm on the whole would appear to be a secondary form of adjustment rather than a primary,[5] although it must be admitted that in operations on aneurysms where the vessels have been directly handled severe arterial vasospasm has been clearly observed, and in turn lessened by the application of local vasodilators such as papaverine. Circulatory improvement following active decompressive measures is probably due to the re-establishment of a favourable balance between the existing arterial blood pressure and the pressure outside the arteries, and the consequent reduction in the resistance to the blood flow.

VENOUS CEREBRAL CIRCULATION

Finally I want to review and discuss briefly a perspective of the venous cerebral circulation. The venous circulation is in every way as important as the arterial. The volume of the venous blood lake in the brain is greater at times than the arterial. This fluid volume becomes an important component of the balance in pressure systems throughout the body as a whole, and is influenced by the regular and irregular pulse waves which reach it from outside the head—from the heart, lungs and even the abdomen, and also from pressure changes in the atmospheric medium in which the body finds itself. These pressure waves are modified by physical factors according to the calibre and nature of the channels of communication that exist between the cranium and these outside systems. The venous 'lake' serves to spread, absorb and further modify these pressure fluxes, doing so in conjunction with the cerebrospinal fluid volume, so helping to regulate the phasing and summation of the primary and reactive pressure waves which tend to pass across, or hammer at, or distend the brain tissue and vessel walls which intervene.[6]

Dynamically speaking, the brain can be looked upon as a complexly involuted membrane situated between the various pulsatile fluid systems. Whereas the force of the arterial pulse is translated into linear velocity within its strong muscular channels, this does not happen to the same extent in the veins, where the force tends to dissipate in all directions, so producing a greater immediate effect upon the enveloping brain tissue. Largely through the venous cerebral blood the elastic brain sponge becomes a passively activated 'pump',[6] which in turn becomes an important mechanism for the promotion of linear flow to the cerebrospinal fluid and to the general blood circulation. In conditions where the dynamic equilibrium is upset by disease, either inside or outside the venous system, the phasing of the pulse waves may become such that the passive cerebral pump is rendered neutral or adynamic. This state of affairs can be seen in extremes of either high or low intracranial pressure and where large portions of the brain become impacted by dislocation under a fixed structure like the falx, as may occur in subdural haematomata. If this neutral adynamic state were to exist for any length of time, stasis of the cerebrospinal fluid and the venous and capillary blood would occur, resulting in varying degrees of cerebral ischaemia. If venous hypertension were also present, cerebral oedema would eventually build up.

Again, active measures can break this vicious cycle by the removal of compressing or dislocating lesions, thereby reconstituting the normal tissue resilience. Further, the free flow of venous and cerebrospinal fluids can be regulated by the provision of appropriate communications between one pressure system and another or by a vent to the environmental atmosphere. Raised intracranial pressure is a relatively infrequent accompaniment of arterial hypertension, but it is an almost inevitable result of venous hypertension. In our everyday surgery we associate the occurrence of high cerebrospinal fluid tension with high venous tension. Lowering the one lowers the other, and by controlling these two factors we can limit or prevent the development of malignant forms of oedema—the greatest obstacle in brain operations. It is for these reasons that good anaesthetic skill has become so vital to the success of the surgeon, especially in the prevention of venous congestion.

CONCLUSION

I hope that, by using these examples, I have brought out perspectives and principles which I personally have found useful. They are dynamic complexities which more often than not cannot be easily appreciated. Where we have failed beyond a reasonable measure to control these dynamic complexities, our surgery has remained limited or unsuccessful. Unfortunately we can never quite reconstruct the reasons for our errors, and even at post-mortem examinations, where dynamic influences have ceased to act, they remain for the most part unrecognized. The broken threads of observation keep bearing upon our judgment and intuitive actions more and more as experience grows, giving rise to conceptions which are hard to command at will and still harder to teach. They all refer fundamentally to the cerebral circulation and force us to conceive this in its completely holistic context. It is here, of course, where practice and theory do not easily meet together and where the essential limitations of the surgeon's craft, basically one of tissue reconstruction, are most manifest. Surgical craft is therefore advancing beyond mere reconstruction of tissues to the regulation of intracranial dynamics as a whole. Since the human is the ultimate subject of this dynamic research and its application, and the neurosurgeon is the mediator bound in all his actions by ethical and sociological considerations, the neurological surgeon has taken his place as the ecologist of the nervous system.

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THE MANAGEMENT OF ACUTE POLIOMYELITIS*

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There is no specific treatment for acute anterior poliomyelitis itself, but certain of the urgent complications which endanger life may respond successfully to special skilled care. The methods outlined below are based on the experience gained at the Children's Medical Centre in Boston, Mass., where over 700 cases of acute poliomyelitis were treated during the epidemic of 1955. I became acquainted with these methods during a visit to America in 1957.

The diagnosis of acute poliomyelitis is seldom, if ever, an orthopaedic problem and the treatment during the febrile phase is carried out by the physician or paediatrician, although the orthopaedic surgeon has daily access to all cases even during the period of isolation.

There is no contra-indication to a lumbar puncture if this is necessary in order to make a diagnosis. It is felt very strongly that a member of the orthopaedic staff should see the case before this is done, for lumbar puncture frequently makes subsequent orthopaedic examination very difficult.

An attempt is made to gauge the extent of paralysis and its rapidity of spread without disturbing the patient more than is necessary. Points to be looked for are:

(*a*) Slow progression of paralysis with little change is usually a good sign whereas rapid onset and spread of the paralysis carries a poor prognosis.

(*b*) Difficulty in holding up the head and weakness of the shoulders suggest that respiratory paralysis will occur from involvement of the phrenic roots.

(*c*) If muscle tenderness is present across the lower abdomen and in front of the thigh, paralysis of the whole of that leg is likely to develop.

(*d*) Close observation is kept of the progress, because it is important to know at all times, or to assess as soon as possible in the case of late admission, whether the illness is still in an active phase, in order to predict the outcome of the disease and to anticipate complications.

Paralysis commences 24-36 hours from the onset of the fever and continues for 24-36 hours. Once the temperature settles there is no further spread of paralysis and if the patient has not shown any evidence of grave complications by this time he will not do so in the future. The point to remember is that changes occur with startling rapidity in poliomyelitis and patients cannot be left unwatched; fatal respiratory failure may supervene within minutes. Once the temperature has been normal for 48 hours the danger of complications is well past and the case has entered the convalescent phase where every change is towards recovery.

During the acute phase of the illness, whether or not the patient develops complications, the principles of polio nursing must be enforced to the full. All beds or cribs must have full length boards with firm mattresses. A foot board is essential to help preserve good bed posture and prevent foot drop; a useful modification is the use of canvas tennis boots nailed to the foot board and into which the feet are laced loosely. This is more comfortable than metal bed splints along the whole length of both legs. A bed roll is usually placed under the knees in order to relieve tight hamstrings and a lumbar pillow will keep the back comfortable. Special nursing and posturing ideas have been worked out for the various regions of the body and aid materially in patients' comfort and posture.

With adequate nursing staff and the assistance of physiotherapists in the isolation wards, rigid splinting should be avoided; softer and more pliable posturing aids should be used.

There is no place for formal physiotherapy during the acute febrile stage of poliomyelitis. The physiotherapist under the direction of the orthopaedic surgeon and with the agreement of the physician may move joints passively in order to prevent stiffness. This is governed by the severity of the illness and the pain tolerance of the patient.

For muscle pain hot packs and aspirin appear to be the safest and most effective therapeutic agents. It has been argued that hot packs are not necessary in hot weather but there is no doubt about their value. Hypnotics should be avoided because they may mask premonitory symptoms of disaster.

During the febrile period isolation should be practised, with barrier nursing techniques. The hands of everybody who comes into contact with the patients should be scrubbed with Phisohex and not merely dipped into a basin of disinfectant solution. The best virocidal agent is thought to be tincture of iodine. As has been mentioned above, the period of isolation ends when the temperature has been normal for 48 hours.

It is the consensus of opinion in America that about 20-30% of hospital cases develop some severe complication, either bulbar poliomyelitis or respiratory paralysis.

COMPLICATIONS OF POLIOMYELITIS

The complications may be classified as follows:

A. *Bulbar Poliomyelitis*

1. Upper Cranial Nerve group: Paralysis of the 3rd, 4th, 5th, 6th and 7th nerves.
2. Lower Cranial Nerve group: Paralysis of the 9th, 10th, 11th and 12th nerves.
3. Respiratory Centre group
4. Circulatory Centre group } Respiratory insufficiency.
5. Encephalitic group

B. *High Cervical Cord Involvement*

6. Respiratory paralysis.

The above classification is merely a guide to the various types that may be encountered but it should be remembered that mixed types occur commonly. When this happens the treatment indicated for each type should be given.

Cranial Nerve Groups

1. *Upper Cranial Nerve group.* This involves only the facial muscles and the muscles of mastication. No difficult problems of management emerge. The prognosis is good and recovery is the rule.

2. *Lower Cranial Nerve group.* This is more severe because the muscles of deglutition are affected. The patient is unable to swallow and may aspirate food or secretions. Laryngeal paralysis with adduction of the vocal cords may obstruct the airway completely. These patients are terrified and require very careful handling.

Treatment. Constant suction and postural drainage are carried out even in a tank respirator. If secretions cannot be

* Based on a paper presented at the South African Medical Congress, Durban, September 1957.

controlled conservatively, then a tracheotomy is necessary. An important early decision is whether a tracheotomy will be required so that it may be done as an elective procedure, under an anaesthetic, and not as a desperate emergency on a suffocating patient. Absolute indications for tracheotomy are vocal-cord paralysis and infra-laryngeal paralysis resulting in elevation of the larynx against the base of the tongue.

Respiration through the tracheotomy tube can be spontaneous, by means of an intermittent positive pressure respiratory aid (I.P.P.R.) or pressure cycled i.e. with a tank respirator. In all cases the air must be humidified to prevent caking of the secretions in the trachea.

3. *Respiratory Centre Involvement*

This type is usually associated with involvement of the circulatory centre, which further endangers life. The patients are restless, apprehensive and cyanosed and they breathe irregularly with long periods of apnoea due to impaired discharge of stimuli from the respiratory centre. The condition has been likened to a 'fibrillation' of the centre.

Treatment. These patients fare badly in a tank respirator as they are unable to breathe in time with the machine, owing to the irregular outflow of stimuli from the respiratory centre. In fact a respirator will probably kill them. Attempts have been made to knock out the centre with large doses of morphia so that they will not fight the machine but these attempts have been unsuccessful. The patients require oxygen therapy *via* an intra-nasal catheter or by means of a tracheotomy if that is indicated. A regular breathing rhythm should be assured by means of an electro-phrenic stimulator, which blocks off all the irregular and ineffectual discharges from above. A tracheotomy may help here by eliminating much of the dead-space air and decreasing respiratory resistance. Prognosis in this type of case is very bad. Most cases die with pneumonitis and pulmonary oedema despite antibiotic 'cover'.

4. *Circulatory Centre Involvement*

These patients have a florid, dusky look and show evidence of cyanosis. They sweat profusely and are restless and develop hypertension up to about 200 mm. Hg with a low pulse pressure and hyperpyrexia to 106°F. One can do nothing to control their condition. They collapse suddenly and go into a state of shock. Practically all cases die and at autopsy are found to have had very severe haemorrhagic pneumonitis and pulmonary oedema.

5. *Encephalitic Group*

These patients show personality changes. They are very confused and suspicious and think the doctor intends to harm them. They complain of very severe headaches and stiff necks and backs, and commonly develop tremors and convulsions. Respiration becomes shallow and irregular, with periods of apnoea. It may be very difficult to differentiate these cases from those having respiratory-centre involvement, but it is important to do so because the treatment is different. If they respond to oxygen therapy, they are respiratory-centre cases. In the worst cases the temperature continues to rise, they become stuperose and comatose, and they die in convulsions.

Treatment. For respiratory insufficiency a tank respirator is the only thing that will save these patients. For the rest they require very careful nursing and sedation but this must be given with great care, for they can be knocked out quite suddenly. Prognosis in these cases is bad but certainly better than in the respiratory-centre group.

6. *Respiratory Paralysis*

Patients with this type of involvement can be differentiated from true bulbar polio by testing the function of the diaphragm and intercostals. If asked to whistle or blow against one's finger or to take a deep breath and count, the weakness is fairly obvious. If observed from the beginning, spread of weakness to the neck and shoulder girdle musculature will often give warning of a respiratory paralysis.

TREATMENT OF RESPIRATORY PARALYSIS

This consists of artificial respiration and it should be applied early, before the patient is in desperate need of air. Only a tank respirator is used in the acute phase, all other respiratory aids being of use only in the later phases of the disease. A working knowledge of the physiology of respiration is essential for full appreciation of the treatment of respiratory paralysis. Space forbids an explanation at this stage but many standard texts are available, or an anaesthetist colleague may be consulted.

Indications for the Use of a Tank Respirator

1. When a patient with some respiratory involvement is admitted in an exhausted state, as is often the case after a long journey by ambulance, he should be put straight into a respirator for a rest. Such a patient will often go straight off to sleep and on awakening will feel much better. He can then be taken out.

2. When the vital capacity is 50% of the predicted normal the patient should go into the respirator for a trial or for rest periods, and should sleep in the respirator during the early stages of the disease to avoid the swift and silent approach of disaster when no warning is possible.

3. When the vital capacity is reduced to 33% of the predicted normal the patient should remain in the respirator until he recovers a safe tidal volume.

Being put into a respirator can be a very frightening experience and psychological preparation is most important.

Methods of Assessing the Need for Artificial Respiration

Arythmia of the respiratory centre or incoordination of muscular activity do not of themselves require artificial respiration. The only absolute indication is hypoventilation and the only exact method of testing this is by alveolar air estimations and arterial oxygen and carbon dioxide saturation curves. These techniques must be carried out in a laboratory and are not practicable in the severely ill patient.

In practice one measures the vital capacity with a spirometer and estimates its efficiency as a percentage of a predicted figure. A rough estimate of the predicted vital capacity can be made from the following table:

	5-15 *years*	*Over* 15 *years*
Males ..	250 c.c. per year of age	25 c.c. per cm. of height
Females ..	200 c.c. per year of age	20 c.c. per cm. of height

The results obtained by this method will be an adequate guide to whether a patient requires respiratory assistance. Once it has been decided that the use of a tank respirator is necessary the required tidal volume can be estimated accurately from Radford's nomogram[1] (Fig. 1) and the settings of the machine can be adjusted accordingly. Finer adjustments can be made after accurate biochemical analysis, if this appears necessary. The whole purpose of artificial respiration is to

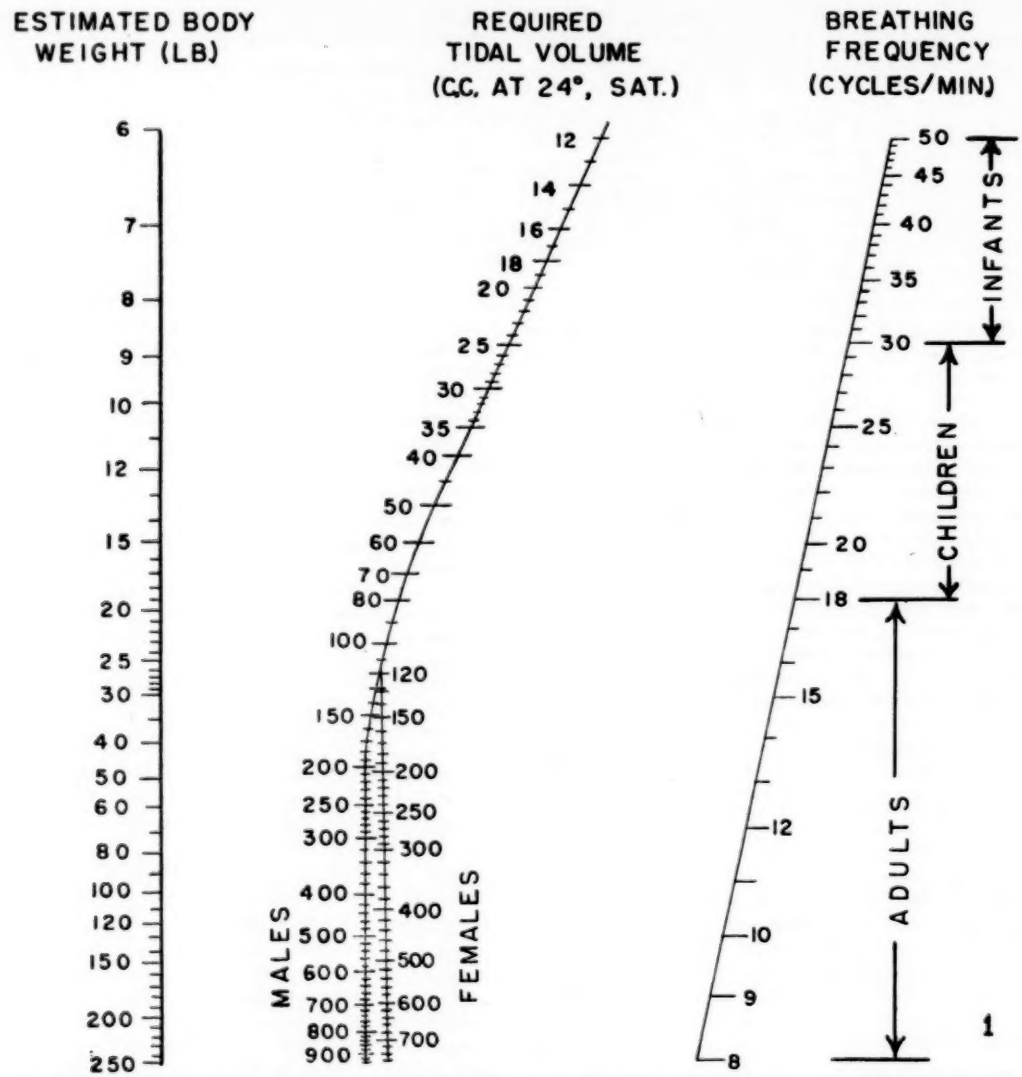

Fig. 1. Ventilation nomogram to be used in estimating the adequacy of tank respirator settings (after Edward P. Radford, Jr., M.D., Department of Physiology, Harvard School of Public Health).

Determine the required tidal volume from the nomogram plus corrections; then adjust tank pressure or frequency until the average measured tidal volume equals the predicted value.

Correction factors to be applied to tidal volumes obtained from nomogram:
Daily activity and eating: Add 10%
Fever: Add 5% for each °F above 99° (rectal)
Altitude: Add 5% for each 2,000 feet above sea level.
Tracheotomy: After all above corrections have been added, subtract a volume equal to ½ the body weight.

get rid of the excess CO_2 in the blood, the hypoxia being of secondary importance. CO_2 retention causes acidosis and upsets the whole electrolyte balance, but once the CO_2 level is controlled the electrolytes assume their normal proportions, although some initial assistance may be necessary.

General Measures

All patients with respiratory embarrassment should receive antibiotics in order to prevent the development of pneumonia. When necessary, the electrolyte balance must be adjusted with whatever ions are indicated. The importance of posturing and the control of secretions have been stressed. The general nutritional state should not be allowed to lapse too far.

Emergency Measures and Equipment

It is essential that everyone who works with acute poliomyelitis patients should be able to recognize the warning signs of complications and trained to act accordingly. They

must all understand the working of tank respirators and be capable of using an emergency hand bellows if necessary.

The following items of equipment should always be available in the ward and in full working condition. Under no circumstances should 'borrowing' be allowed:

(1) Tank respirator, (2) bellows resuscitator, (3) tracheotomy set, (4) bronchoscopy set, (5) suction machine, (6) laryngoscope tray with airways and endotracheal tubes, (7) oxygen, (8) a spirometer with disposable mouthpieces, and (9) an electro-phrenic stimulator. Furthermore, when acute cases are in the ward a doctor should always be *within call*—being *on call* is not sufficient, because minutes may make all the difference.

SUMMARY

Methods of handling cases of acute poliomyelitis are described.

The complications of bulbar poliomyelitis are classified and the essentials of treatment outlined.

The indications for artificial respiration are given and methods of assessing the predicted vital capacity and required tidal volume are shown. Radford's nomogram is reproduced.

Emergency equipment is listed.

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VIEWS ON THE TREATMENT OF CARCINOMA OF THE BREAST *

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My remarks in this paper are addressed, in the first place, to the average general surgeon—amongst whom I class myself—practising under present South African conditions. Because we are not mammary specialists, we cannot depend on our personal experiences alone, and must, to a certain extent, be guided by the collective experience of surgeons working in the more populous areas of the world. At the same time we must realize the particular conditions under which we practise, and adjust our treatment to suit them.

Unfortunately the masses of statistics with which the surgeon is confronted, with all their contradictions—probably more apparent than real—are difficult to digest. They have added to the headaches of the surgeon when, *in a given patient*, he has to decide on the 'best' method of approach. Moreover, his confidence has been somewhat shaken by Harrison's recently published statistics,[1] according to which it is *not possible to prove that the final mortality rate from carcinoma of the breast is actually influenced by treatment at all.*

I am first going to discuss the problem from a theoretical point of view; and then I shall outline an approach which is practicable and, I trust, in keeping with present knowledge. I do not believe that the bulk of surgeons practising in 1958 should adopt procedures which, if acceptable at all, can only become generally acceptable in, say, 1968. Of course, it is a completely different matter for a few fortunate individuals who have the material and all the facilities for clinical experimentation and control. Generally speaking, it is wiser to wait until we can be guided by the results obtained in large and comparable series of cases observed over long periods in the same institution. As far as I can judge, at the present time there is as little cause for over-optimism about the newer methods of treatment as there is for undue pessimism about the older methods when, that is, we consider the problem merely from the point of view of the survival rate. In the field of palliative treatment, however, undoubted advances have been made.

THEORETICAL CONSIDERATIONS

1. At its inception carcinoma of the breast is *not* a generalized disease. I make no apologies for this statement, because in my opinion it is a timely one. There is a time when carcinoma of the breast is a local disease, *and therefore curable*. Neither has it been proved to be a widely disseminated disease in a significant percentage of patients at the time of its first diagnosis. For a certain period, therefore—and this may be only a few months or several years—the disease is sufficiently localized to enable us to eradicate it by treatment applied merely to those tissues which the surgeon is able to excise with a minimum of danger. These tissues are the mammary organ and the axillary lymph nodes (I discuss other lymph nodes below).

2. *An immunity response* to the invasion by malignant disease is a theorectical consideration which cannot be disregarded. A natural cure has been reported in many cases of proved carcinoma of the breast. In other cases cures have followed on treatment which certainly could not have eradicated all cancerous tissue. Malignant cells have been found to enter the bloodstream at a relatively early stage of the disease;[2] but not all patients so affected succumb to the disease. Somehow and somewhere these cells are destroyed. Destruction most likely depends on the presence of antibodies which develop in the blood and other body fluids and tissue cells. There may even be a hormonal participation in this immunity response. In this respect it is well to remember that the cancerous process is almost certainly an irritating one to the tissues of the host. How can we otherwise explain the development of so much fibrous tissue? The laying down of collagen fibres is an important defensive mechanism which is found characteristically in the so-called atrophic scirrhous carcinoma. Malignant cells spilt at operation may become encapsulated by fibrous tissue and in this way be put out of action. In summary I emphasize that on at least two theoretical grounds it would be a mistake to accept the pessimistic view that no matter what we do, or omit to do, the end result will be just the same.

A recent publication by Berkson *et al.*[3] was very welcome, because it showed us the other side of the picture. They reviewed the results obtained in nearly 10,000 patients treated at the Mayo Clinic over a period of 45 years from 1910 to 1954. I want to refer particularly to the 660 patients who received treatment in the years 1910 to 1915, i.e. over 40 years ago, and of whom an up-to-date record is available. Ten years after treatment 38·1 out of every 100 of these patients were still alive, as compared with 80·3 out of every

* A lecture delivered at the 1st congress of the South African Association of Surgeons, Cape Town, April 1958.

100 of the normal population. On this basis some 47% of cases of breast cancer, whether early or advanced, might be alive 10 years after their treatment. In patients whose axillary nodes were free from metastases at the time of operation, the 10-year survival rate was 75%. After the tenth post-operative year the death rate in patients who had carcinoma of the breast ran almost parallel with that of the general population.

AN OUTLINE OF TREATMENT

In discussing treatment in detail, I shall do it under rather unorthodox headings. Nevertheless, I do so in order really to be orthodox and not to be misunderstood.

1. *The Very Hopeful Case*

This commonly is the early, but not necessarily the very earliest case. In this patient the axillary nodes are still free from metastases, and the tumour is small and mobile, and is preferably placed in the lateral half of the breast. The fact that it has been there for several years is of less importance than the fact *that the axilla is not* yet involved. During the first few months a short history is actually indicative of a worse prognosis than a somewhat longer history. The explanation of this paradox lies in the fact that the patient seeks advice earlier, because the symptoms in her case are more pronounced; and the symptoms are more pronounced because the tumour is of a more active nature, a fact which is likely to become apparent on histological examination after the operation or at biopsy.

There is no proof that the youth of the patient influences the prognosis. Factors which do influence the prognosis are the anatomical situation, pregnancy and lactation, and a histologically active type of cell.

Radical mastectomy is the treatment of choice during this stage and for this type of case. But I must emphasize that the mastectomy must be a truly *radical* one. I am adamant about the principle that the earlier the malignancy, the more radical the excision must be, for it is in this type of case that we can expect a complete cure. It is here, much more than in the advanced case, that half measures may lead to failure in a patient who almost certainly could have been cured if we had not allowed ourselves to be influenced by the apparent infancy of the growth, and so adopted a less radical method. In my experience a local mastectomy is an incomplete operation.

Once histological examination has confirmed that the lymph nodes are free from disease radiotherapy need not be considered. Its application now is not only unnecessary, it may be detrimental. Irradiation is reserved for a much later period when, in a small percentage of cases, metastases may appear in the skin or elsewhere. The internal mammary nodes, however, may be irradiated in early cases if the carcinoma is located close to the sternum.

Ovariectomy should not be done now. I do not criticize those who use it at this stage, because on theoretical grounds it may well be that ovariectomy in the menstruating female will so change the immunity response to dispersed cancer cells that their activity is significantly subdued. But there is as yet no proof that the development of secondaries will be prevented.[4]

The baneful psychological and physical effects of ovariectomy should be avoided; its beneficial effects must be reserved for such of these cases as may eventually develop secondary lesions.

2. *Where a Mobile Tumour and Enlarged, but Mobile, Axillary Nodes are Present*

The prognosis has now deteriorated, but life expectancy is still 61·3% in 5 years' time, 42·7% in 10 years' and 31·1% in 15 years'. It is therefore obvious that we must still aim at complete eradication of the disease. With few exceptions it is universally agreed that this can be best achieved by a combination of surgical and radiological treatment. But there is some difference of opinion whether the operation should now be reduced to a *local* mastectomy, supported by irradiation of the axillary and other nodes, or whether it should still be a *radical* one supported by radiological treatment. There is also a difference of opinion whether radiological treatment should precede the operation or follow it. I firmly believe that the operation at this stage must still be a radical one. I do so because I have yet to see an axillary recurrence following extirpation of mobile enlarged nodes whether radiological treatment was given or not. I have, however, frequently seen supraclavicular nodes enlarge in these very cases after irridiation of both the axilla and the supraclavicular fossa. It happens in patients who had no palpable nodes above the clavicle before the irradiation. In my experience it is wrong at present to reduce the scope of the operation in favour of radiological treatment.

An eminent radiotherapist, McWhirter,[5] has obtained impressive results by irradiation of the lymph nodes followed by local mastectomy. His results, however, do not equal—insofar as results are comparable—those obtained by the generally accepted methods of treatment.

As far as radiological treatment at this stage is concerned, I believe that it is best confined to the supraclavicular fossa, and the internal mammary area. I do not think it is necessary to irradiate the axilla in cases where the glands were mobile at the time of operation.

I further believe that the chest wall should not be irradiated at all in this type of patient. I must, however, emphasize that my remarks can only apply where the surgeon has done his job thoroughly. If the skin round the tumour is widely excised (disregarding whether a skin graft becomes necessary and the skin flaps are left with a minimum of subcutaneous fat) there need be little fear of local recurrences. A thick pad of subcutaneous tissue, which the radiotherapist prefers, should not be left. If it is, the number of local recurrences will increase. A recurrence in a skin underlain with scar tissue is not a matter of grave concern anyhow. It can be excised. Multiple recurrences can be irradiated. The results are much better where the skin has not been previously irradiated.

It is possible that an even more radical operation may become common practice in the foreseeable future if the results of those who are now practising this method can be proved to be superior to those of the less radical surgeon. In this ultra-radical operation the supraclavicular and the internal mammary nodes are also excised. The argument in favour of this extended operation is a sound one, viz. that in a considerable percentage of the cases where axillary nodes are involved, the internal mammary and the supraclavicular nodes are also involved. Against the extended operation there are two arguments. Firstly it is said that equally good results can be obtained by deep therapy to these two groups of lymph nodes. Secondly it is known that the operative risks are greater when the scope of the operation is thus extended. Although for the present I favour the ordinary radical or

Halsted type of operation, I sincerely welcome the fact that others are performing the ultra-radical one. In time we shall be guided by their results.

Ovariectomy and other forms of hormonal treatment have no place in this stage of the disease.

3. *The Locally Advanced Carcinoma*

I refer to the large, sometimes ulcerated, tumour with fixation to skin and/or deep fascia, and usually with enlarged fixed axillary nodes. This is a radiological problem and the advantages of ionizing radiation must be exploited to their maximum capacity in these cases. In the relatively small percentage of such cases where a previously non-operable tumour retrogresses to operable proportions, I believe one should operate, but not within 3 months after cessation of the radiological treatment. By then full fibrosis will have occurred, and one is not likely to do any harm. Although the standard operation now consists of a local mastectomy, I must admit that not infrequently I enter the axilla. But I do so only after thorough evaluation of the condition as seen on the operating table.

4. *The Widely Disseminated Carcinoma*

At this stage there is no more possibility of a cure being effected and our aim is palliation. The disease may be locally advanced. There may be ulceration, gross oedema, enlarged and fixed nodes in the axilla and the supraclavicular fossa. There may be enlarged lymph nodes in the opposite axilla and secondaries in bone in the chest, abdomen and cranium.

Operation plays no part except perhaps insofar as procedures to relieve pain may be adopted. Irradiation, however, can play an important part by controlling local spread and pain. The radiotherapist refrains from giving maximum doses in these cases. In fact he gives the minimum that will control ulceration, spread and pain. His aim is rather to reserve his treatment and to repeat it as often as is necessary, and wherever a local lesion gets out of hand.

HORMONE THERAPY

Hormone therapy has come to our aid as a powerful weapon in the advanced stages of the disease. Certain breast carcinomas are hormone-dependent, but as yet we have no means of determining whether a particular tumour is so dependent, other than by trial and error. When the tumour is hormone-dependent, the effect of hormone therapy is often profound, and much suffering can be relieved and life considerably prolonged.

Should this powerful weapon not be used at the earliest opportunity? I can only state that I have found no evidence that early hormonal treatment has any influence on the progress of the disease and the prolongation of life. I am aware that some surgeons are employing the hormones much earlier, but until we have the advantage of their results we must follow a course which has by now been reasonably well tested. I must also sound a note of warning and point out that these new agents are potent and potentially dangerous. I have on several occasions been shocked by the speed with which a patient receiving this treatment has been rushed to a fatal end.

A Brief Statement on Hormonal Therapy

The Premenopausal Patient. As soon as it is obvious that the disease can no longer be controlled by irradiation, the first step is to perform double ovariectomy. Within 6-8 weeks we should know whether the effect is going to be satisfactory. If the operation produces no effect, it is unlikely that any further hormonal therapy—medicinal or operative—will be of use. If, on the other hand, the result should have been satisfactory and the disease should again show signs of advance, the next step ought to be hypophysectomy. If, after an interval of retrogression, there is a renewal of advance, androgens should be given. They are best given in the form of testosterone propionate, 150 mg. intramuscularly, twice a week for 10 weeks. Care must be exercised if the blood calcium is raised, if there is renal failure, or if ascites or hydrothorax is present. Unfortunately hypophysectomy is not such a practicable procedure. We, therefore, adopt a course which gives only slightly poorer results, but carries a lower mortality rate. After a successful ovariectomy we wait until deterioration reappears, and then go ahead with androgens. Finally when androgens cannot control the disease any more, an adrenalectomy is performed.

Patients 8 years and more Postmenopausal. Here we depend on oestrogens. Diethyl-stilboestrol, 5 mg. twice daily by mouth, is a satisfactory dose. Larger doses are no more effective, and will merely produce uterine haemorrhage. If oestrogens do not control the disease any longer, hypophysectomy is indicated; but in practice we commonly resort to double ovariectomy plus double adrenalectomy instead.

In spite of the almost miraculous results we obtain now and then, I very much doubt whether adrenalectomy is a procedure which has come to stay. It would be a different matter if we could determine beforehand which patients stood a chance to benefit by the operation. At present—apart from the occasional miracle that may take place—we are subjecting three women to all the pain of surgery and the removal from their family on the eve of their demise with nothing better in view than that one of them may live some 18 months longer. I am limiting the operation to patients with secondaries in skin, pleura and bones, and do not recommend it where metastases are located in the brain, the lungs and the liver. In any case, the decision to do the operation must not be postponed to a time when no more benefit can possibly be expected from it.

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College of Physicians, Surgeons and Gynaecologists of South Africa. Margaret Orford Trust Lecture. Prof. Robert J. Kellar, Professor of Obstetrics and Gynaecology of the University of Edinburgh, will give a lecture, under the aegis of the College, on 26 August 1958 at 8.15 p.m. in the Physiology Lecture Theatre, Medical School, Mowbray, Cape. The subject will be 'Modern Views on Toxaemia of Pregnancy'. All members of the profession are invited.

Kollege van Interniste, Chirurge en Ginekoloë van Suid-Afrika. Margaret Orford Trust Lesing. Prof. Robert J. Kellar, Professor van Verloskunde en Ginekologie, Universiteit van Edinburgh, sal 'n lesing gee, onder beskerming van die Kollege, op 26 Augustus 1958 om 8.15 nm. in die Fisiologie-Lesingsaal, Mediese Skool, Mowbray, Kaap. Die onderwerp sal ‚Modern Views on Toxaemia of Pregnancy' wees. 'n Uitnodiging word aan alle geneeshere gerig om hierdie lesing by te woon.

FAMILIAL PES CAVUS, ABSENT TENDON JERKS AND EXTENSIVE MUSCULAR ATROPHY

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Numerous heredo-familial disorders producing muscle atrophy have been described. Different syndromes present from time to time, each having characteristics which might appear specific for the family concerned. Only those syndromes have survived, in which the defect is slight or in which the defect only becomes severe or manifest after the age of procreation or in those who inherit the condition as a sex-linked genetic factor. An interesting example of this last mode of inheritance was described by Popow,[1] who followed four generations of a family of which 9 males were affected, showing features of the Roussy-Lévy syndrome.

Considering certain heredo-degenerative disorders, Spillane[2] has postulated a spectrum of disorder ranging between affections of the cerebellum and spinal cord, of which Friedreich's disease and peroneal muscular atrophy are examples, with the Roussy-Lévy syndrome as a sub-group. Different families have features which tend to be specific. This concept may be extended to include the higher centres, e.g. cases of mental disorder occurring in families with peroneal muscular atrophy, as described by Gibson,[3] or with peripheral nerve affections, as in hypertrophic polyneuritis of Déjerine and Sottas or as in peroneal atrophy itself. Concepts of this nature, however, over-simplify the problem. The term degeneration is all-embracing and does not take into account the possible varieties of metabolic and structural change which produces the same end-result. Metabolic failure at different stages of the same process or of different processes might be genetically determined and account for the variety of manifestations.

Peroneal muscular atrophy was first adequately described in France by Charcot and Marie,[4] and in England by Tooth.[5] The condition is transmitted as a Mendelian dominant, as a recessive, or rarely as a sex-linked gene. The onset is usually in adolescence but ranges from infancy to early adulthood. As is so often the case, the original description bears only a partial resemblance to the condition as we know it today. The original papers of Charcot and Marie and of Tooth, as summarized by Symonds and Shaw,[6] made no mention of pes cavus and only brief incomplete references to the reflexes. The condition was described as a progressive atrophy beginning in the feet and legs and affecting the hands and forearms after a lapse of several years. Sachs[7] was probably the first to emphasize the association with bilateral pes cavus. Sensation was generally described as normal. Since then, however, occasional cases have been noted with sensory disturbances, the commonest being loss of vibration sense in the lower extremity, other modalities being rarely affected. England and Denny-Brown[8] described 7 generations of a family consisting of 303 members, of which they studied 18. Two of these had extensive sensory trophic changes in addition to the muscular atrophy.

Roussy and Lévy[9] published their findings in a family showing bilateral pes cavus, weakness of the hands, and total absence of tendon jerks. They regarded the condition as a separate entity, distinct from Friedreich's disease on the one hand, and from peroneal muscular atrophy on the other. Symonds and Shaw[6] in the same year published a report of a family with a similar picture and considered that both families represented *formes frustes* of the Charcot-Marie-Tooth disease.

A strong case can be made for the Roussy-Lévy syndrome as a separate entity; it has possibly as much right to separate identity as Friedreich's disease or Charcot-Marie-Tooth disease, all showing some features in common—for example, peroneal muscle atrophy with cerebellar signs of Friedreich's ataxia, as described by Ross[10] and Greenfield.[11] Of particular interest is the family described by Spillane.[2] Of the 21 affected members, 16 showed the Roussy-Lévy syndrome, 4 of whom had in addition an explosive type of dysarthia; 2 and possibly a 3rd, showed typical peroneal muscular atrophy. One showed typical Friedreich's disease with kyphoscoliosis, bilateral pes cavus, absent knee and ankle jerks, and bilateral extensor plantar responses. Von Bogaert and Borremans[12] presented 6 cases resembling the Roussy-Lévy syndrome, kyphoscoliosis, occurring in 4, lordosis in 1, and mental retardation in 1. There was no evidence of sensory or cerebellar disturbances. Rather than group their cases under Friedreich's disease, they postulated a separate genetic kyphoscoliotic factor. Other points of distinction of the Roussy-Lévy syndrome are the extreme chronicity of the condition, the widespread nature of the disorder, even though previously this may have been confined mainly to areflexia, and the absence of the cerebellar, ocular and cardiac involvement in cases of Friedreich's disease.

THE AFFECTED FAMILY

The family described here was discovered when the patient A.F.A. presented at the medical out-patients department of the Johannesburg General Hospital in order to obtain a medical certificate for disability pension. The family tree is shown in Fig. 1, in which the members with pes cavus and

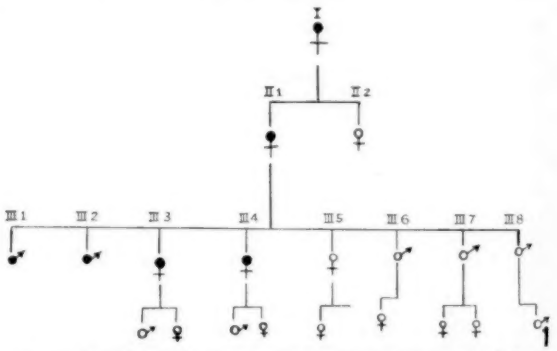

Fig. 1. Inheritance shown through 4 generations. Black circles denote affected members.

absent tendon jerks, and for muscular atrophy, are indicated with black circles. It will be seen that one-half of the members of the 3rd generation shown are thus affected.

1 (III 1).

A.F.A., aged 39 years, married, had no children of his own. He complained of progressive weakness of the whole body. The trouble first started in 1939 when, at the age of 21, he developed

weak ankles so that his feet would turn inwards. At this time he had already noted the development of 'hammer toes'. The condition slowly progressed, so that in 1942 an arthrodesis was performed on both ankle joints, with marked improvement in walking. Weakness of the hands was noted first about 10 years later, and has progressed to the present stage, when opposition of the thumbs is impossible. Weakness of the shoulders and difficulty in rising from the sitting position have been noted lately. There has never been any sphincter disturbance. Eyesight is normal and the patient walks well with a narrow high-stepping gait. When questioned about other members of the family, he stated originally that they were all normal, but at a subsequent interview numerous abnormalities were disclosed.

On examination: A well adjusted male of good intellect. B.P. 120/70 mm. Hg. Normal cardio-vascular, respiratory, gastro-intestinal and genito-urinary systems. The cranial nerves were intact, with the possible exception of the muscles of expression. There was a little difficulty in whistling, and the smile was rather straight, suggesting some weakness of the zygomaticus. The blown-out cheeks were of poor tension and the temporal muscles were small. The muscles of the shoulder girdle, pelvic girdle, hands, feet and legs were small and atrophic. Over the gluteus maximus and medius fibrillation could be seen. The legs show the typical 'rooster leg' appearance of peroneal-muscle atrophy and the feet show marked pes cavus with the typical 'Friedreich's toes'. The tendon reflexes were absent; the cutaneous reflexes were present and normal. Sensation was intact with the exception of vibration sense, which was lost in the lower limbs up to the hips. Muscle tone was diminished and movement and power in the affected muscles was reduced. Cooordination was normal; cerebellum normal.

Facial muscles: Slight weakness of zygomaticus and orbicularus oris on both sides.

Shoulder girdle: Moderate atrophy of all muscle, more marked on the right side, where complete atrophy of rhomboids.

Pelvic girdle: Atrophy of all muscle groups on both sides, most marked in the glutei.

Lower extremity: Moderate atrophy of all muscle groups on both sides. Marked atrophy of leg muscles and small muscles of feet.

Upper extremity: Moderate atrophy of all muscles on both sides, with marked atrophy of small muscles of hand and total loss of opponens pollicis.

Spinal, thoracic and abdominal musculature: Power slightly reduced on both sides; otherwise normal

Muscle reaction to stimulation showed the reaction of degeneration over affected muscles. Muscle biopsy confirmed the atrophy. Details of muscle atrophy are as follows: The 24-hour excretion of creatinine and creatine showed an increase in creatine to 326 mg. per 24 hours (average). All other investigations were normal, with the exception of the *CSF* protein, which was 80 and 84 mg. % on 2 occasions.

The progression of the condition over the years has been extremely slow. The patient is a carpenter and is still able to supplement his pension by doing odd jobs.

2 (II 1)

Mother of case 1, aged 68 years. Has pes cavus, broad feet, absent knee and ankle jerks, and loss of vibration sense up to the knee Cerebellum normal on testing. States that she developed 'hammer toes' at about 16 years of age, and that her mother had claw toes with high arched feet and had to wear boots for as long as she could remember. The family originated in Germany. She has a sister who cannot be traced.

3 (III 3)

Sister of case 1, aged 44 years. Has pes cavus, broad feet, absent ankle jerks, intact sensation and no cerebellar signs. Her son of 18 years and daughter of 14 years are both said to be normal.

4 (III 5)

Sister of case 1, aged 40 years. Has high arched feet but no pes cavus. Reflexes extremely brisk, particularly in the lower limb, plantars flexor. Sensation intact. No cerebellar signs. Has son aged 7 years, has high arched feet, otherwise normal; daughter aged 9 years, normal.

5 *and* 6 (III 6 *and* III 7)

Twin brothers of case 1, aged 35 years. High arched feet, no pes cavus. Reflexes and sensation normal. No cerebellar signs. One has a daughter of 7 years, normal, and the other has daughters aged 5 years and 3 years, both normal.

7 (III 4)

Sister of case 1, aged 31 years. Has high arched broad feet, legs thin up to knees, and arms thin up to elbows, and has noted that she has been getting thinner and weaker progressively over the past 10 years. Wasted thenar eminences; atrophy of muscle of shoulder girdle most marked on the right. Details of muscle atrophy are as follows:

Facial muscles: Normal on both sides.

Shoulder girdle: Slight atrophy of all musculature, most marked on the right side, where mainly in rhomboid.

Pelvic girdle: Normal on both sides.

Lower extremity: On both sides slight atrophy of all groups and moderate atrophy of leg muscles (except peronei and long extensors and flexors, which are normal).

Upper extremity: Slight wasting of all muscles on both sides. Moderate atrophy of small muscles of hand.

Absent reflexes. Sensation normal, cerebellum normal. Has 2 children aged 3 years and 3 months respectively, both normal.

8 (III 8)

Brother of case 1, aged 28 years. High arches; normal toes; marked hyperreflexia, expecially in the lower limbs. Sensation normal. Normal cerebellum. His son aged 2 years is normal.

9 (III 2)

Brother of case 1, aged 24 years, pes cavus, absent ankle jerks, sensation normal. No cerebellar signs.

DISCUSSION

In Friedreich's ataxia, peroneal atrophy and the Roussy-Lévy syndrome, the one frequent feature which is common to all is pes cavus. Superficially, one might regard this as a link between the three conditions, but it must be remembered that the pes cavus is a secondary manifestation and is not itself genetically determined, the primary defect occurring either in the peripheral nerves or anterior horn cells of the spinal cord and resulting in atrophy of the small muscles of the foot. The unopposed action of the long muscles of the leg, if not affected at the same time, or if affected to a lesser degree, moulds the foot into the characteristic deformity. The long muscles of the legs may be affected first or more severely, and then pes cavus does not occur; in fact pes planus may result. Peroneal atrophy is a bad term, because the brunt of the atrophy may, as previously mentioned, occur in other muscles such as those of the foot, and particularly those of the anterior compartment of the leg, leading to foot drop. When in fact the major atrophy occurs in the peroneal group, the patient stumbles with the foot in an inverted position.

In peroneal atrophy, the site of the primary pathology has been a source of contention for years. In the original description, Tooth held that the pathology was primarily in the peripheral nerve, as opposed to Charcot and Marie, who cited the spinal cord. This argument has continued to the present time, with Kinnear-Wilson[13] in favour of the spinal cord, and England and Denny-Brown[8] in favour of the peripheral nerve and regarding the spinal change as secondary to peripheral degeneration. The latter authors consider the Roussy-Lévy syndrome a separate entity with primary spinal pathology.

Case 1 has the typical 'rooster legs', pes cavus, atrophy of the small muscles of hands starting years later (involving to a lesser extent the forearms), loss of vibration sense up to the hip joint, all of which are characteristic of peroneal muscular atrophy. But against this diagnosis is the fact that the wasting does not stop at the lower third of the thigh, nor does it stop at the elbows. Marked wasting is noted with fibrillation affecting the shoulder and pe vic girdle.

The slight atrophy of the muscle of the face, with atrophy

of the shoulder girdle, could be confused with the muscular dystrophy of Erb or with that of Landouzy and Déjerine, but this latter condition is excluded clinically by the involvement of small muscles, pes cavus, areflexia, etc.

Friedreich's ataxia can hardly be included in the differential diagnosis, the only compatible features being the pes cavus and areflexia of lower limbs.

Case 1, and to a lesser extent case 7, are considered to be examples of the Roussy-Lévy syndrome, case 1 showing a degree of atrophy hitherto not recorded. Gordon[14] reported 4 cases of the Roussy-Lévy syndrome with right-sided scapulo-humeral atrophy; in the present cases scapulo-humeral atrophy was most marked on the right side. In the present family the onset of the disease occurs in early adult life, with gradual development of pes cavus, and in most members this remains the only manifestation. The progress is extremely slow and the atrophy out of all proportion to the disability produced. With the aid of well-timed orthopaedic procedures, these people can be kept active for what might well be a normal life span.

SUMMARY AND CONCLUSIONS

Four generations of a family comprising 21 members (Fig. 1) have been studied. A hereditary neurological disorder was observed. The mode of transmission appears to be through a Mendelian dominant gene, for the incidence of abnormality in the 3rd generation approaches the anticipated 50%.

Widespread atrophy is noted in one member and to a lesser degree in another. No evidence of cerebellar involvement could be found. An unusual degree of hyperflexia was noted in 2 other members. The 4th generation were observed to be quite normal at present, but affected members are to be anticipated as the condition in this family only becomes manifest in early adult life. The condition is considered to be an example of the Roussy-Lévy syndrome. Its relation to Friedreich's ataxia and peroneal muscular atrophy have been discussed, the latter in detail, as the Roussy-Lévy syndrome is usually included under this heading.

A plea is made for the retention of the syndrome as a separate entity.

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SOME ASPECTS OF POSTGRADUATE SURGICAL EDUCATION IN THE UNITED STATES OF AMERICA AND SOUTH AFRICA*

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Having recently returned to Johannesburg after 1 year in the United States, where I had excellent opportunities to note the trends of postgraduate and undergraduate education, I feel that there are aspects of their training programmes that could be utilized with profit by South African schools. After 14 years of personal experience in full-time academic surgery I feel reasonably qualified to offer comments in this field. Professor Churchill, a doyen in the world of surgery and medical education, states: 'The days are past when a great consultant and a great professor are synonymous. Today they are two different careers and not competing with one another. A great consultant is usually not a great teacher and *vice versa*. The ancilliary advances in surgery, standard techniques, antibiotics and post-operative care have made it safe for the patient to be operated on by young trainee surgeons under supervision. There is no question that the senior surgeons of an older school are good and able men but they cannot encompass the modern scene as clearly as younger surgeons trained in a different era.'

The following lengthy quotation is from a report to the American Surgical Association by the special committee on Graduate Surgical Education, April 1953, of which Prof. Oliver Cope, of Harvard University, was the chairman:

'Criticisms of present-day graduate teaching are numerous. The most obvious is that too few surgeons are really interested in anything but the technical training . . . . Such narrowness of interest in the teaching stems, of course, directly from the narrowness of the concept of patient care. The interest of too many surgeons flags the moment the incision is closed. It is a rare surgeon whose understanding of fluid balance, nutrition, aetiology and preventive medicine of the diseases of his own interest carries the same stamp of authority as his technical competence. So, too, for the social aspects of patient care. Too many surgeons pride themselves on the narrowness of their knowledge and technical capacity. It is as if they thought ignorance outside their sphere indicates greater knowledge within. Such pride is absurd when it is realized how broad and intricate medicine is. What is worse, it smacks of irresponsibility.

'It is also a fair criticism that too few surgical teachers appreciate curiosity. Research is something effete and for the physician . . . .

'Careers in teaching at the graduate level should be fostered as much as in the undergraduate education of the medical student. Universities must share responsibility with hospitals. Older men must be generous in affording opportunities to the younger . . . . . The older surgeon must stop deprecating research in fundamental medicine . . . .

'Whether prolonged graduated residency (registrarship) or apprentice system is practised, what counts is the atmosphere. Education is best carried out in an atmosphere which is intellectually demanding . . . . The surgeon must be emotionally secure enough to match his wits with his colleagues. Instead of ridiculing and criticizing their shortcomings, he must support vigorous departments of medicine, psychiatry and the laboratory services. A free exchange of thought is to be encouraged by combined clinics and frequent consultations . . . .

'A common criticism of many graduate programmes is that the hospital and the staff make use of the intern and resident rather than contribute to their education. Where the practical day-to-day care of patients involves several medical men at levels from students to senior surgeon, it is often difficult to strike a reasonable balance between usefulness and learning. Both are often involved at the same moment. The balance struck depends upon the true interest of the staff in teaching.'

The Committee concluded its deliberations by acknowledging that the influence of the American Boards and the College of Surgeons has been of the greatest benefit because organization of good graduate training programmes followed the stipulations laid down by these authorities. The broad scientific nature of the examinations given by the American Board of Surgery had injected an educational aspect into the programmes. However,

* Based on an address delivered to the Medical Graduate Association, Johannesburg, 30 January 1958, and submitted for publication on 19 March 1958.

they deprecated narrow and extreme specialization. They learned much from examining the procedures of the Royal College of Physicians and Surgeons of Canada. 'A 5-year curriculum of graduate education and training leads to the examinations for Fellowship in Surgery or in a speciality. A broad education is demanded before qualifying. The examinations are rigorous and include examinations in the medical sciences'.

No doubt the new College of Physicians, Surgeons and Gynaecologists of South Africa has learnt much from the Canadian system in contrast with the English or Scottish systems. These latter UK examining bodies do not demand a preliminary approved 4- or 5-year graduated registrarship and it is conceivable that many examinees for these higher diplomata may have never performed even a small variety of major operations!

Naturally, the necessity for a 4- or 5-year training programme is not merely for the acquirement of surgical technique but also to cater for the individual who has research ideas. Prof. Owen Wangensteen, of the University of Minnesota, states: 'The most fundamental requisite of a research project is an idea. A disciplined imagination is at the bottom of every great discovery. The person professing to want to do some research must be looking for something . . . . Persons with ideas may lack intimate knowledge of methods, tools, or techniques by which to undertake the solution of a problem. And frequently, too, persons who have an intimate acquaintance or mastery of techniques are devoid of ideas. Obviously, therefore, for the successful prosecution of research, a combination of talents is necessary, in which a fusion of effort with others gives an accelerated momentum to the project. No one was ever great by imitation. The touchstone of the scientific method is the universal validity of its results. It establishes a finality of proof and agreement which puts aside all speculative rationalization. Such is the superiority of the experimental method over logic.'

A great deal of the progress in the medical and surgical world is due to preliminary research in the laboratory and its application in the clinical wards. This obviously requires a closer liaison between the clinical man and the laboratory man. This link is provided by the registrars, who should have easy access to both. In the Witwatersrand medical school we are slowly understanding the need to integrate our teaching and practice by increasing our cooperation with the basic science departments. Instead of relying wholly on small unit staff meetings we hope that all of us will realize that it is necessary for the whole surgical division to meet more frequently in order to discuss unit records, mortality, morbidity, interesting cases, research results and trends, and organize grand surgical rounds for the benefit of all graduates and students.

HISTORICAL

A brief historical survey of the state of affairs in the United States may be of interest. In 1870, although there were about 20 university medical schools in Germany, there were no medical schools of university rank in America. In 1878, Dr. William Stewart Halsted, a graduate of Yale and Columbia Universities, then aged 26, went to Europe for 2 years of study. The greater part of his time was spent in Vienna, Leipzig, Würzburg and Halle, and he was most influenced by the teachings of Volkmann, Billroth, Thiersch, Bergmann and Mickulicz. As Professor Blalock expressed it, 'These were the golden days in medicine. Bacteriology was dawning, embryology and histology were developing, pathological anatomy was being studied with great vigour and the teachings of Lister were being accepted in Europe. These 2 years had a profound influence on the future of Halsted who was aware of the many deficiencies in the medical schools and hospitals of America. Among other benefits this period of freedom from clinical duties allowed time for study and thought'.

In 1889, the Johns Hopkins University and Hospital were completed. The dean and professor of pathology was Dr. William H. Welch; Osler was head of the department of medicine and Kelly of gynaecology. The professorship in surgery was offered to Sir William Macewen, of Glasgow, but it was refused and Halsted was appointed for 1 year on an 'acting' basis as surgeon to the hospital. When 2 years later he was appointed professor at the age of 39, Welch said that 'no greater good fortune could have befallen the Johns Hopkins Hospital than to have Halsted as Surgeon-in-Chief. He was also instrumental in developing the full-time residency training programme which later proved to be a superb system of postgraduate medical education. He created the first genuine school of surgery in America'. The two principal features of this residency system were (1) a close blending of the work of the basic sciences and the clinic, and (2) a prolonged postgraduate training in which the best candidates were retained for a term of several years. The Halsted modification of the German system consisted in the main in the concentration of responsibility and authority in the resident (or registrar) rather than in the Geheimrat (or chief). In 13 years Halsted had trained such men as Cushing, Bloodgood, Mitchell and others. Among his group of 238 resident surgeons there have been 37 professors, 14 clinical professors, 18 associate professors, 14 clinical associate professors, 17 assistant professors, 16 clinical assistant professors and 23 instructors; the remaining 99 were private surgeons.

MASSACHUSETTS GENERAL HOSPITAL

In 1938 the American Board of Surgery established a 5-6 year period of graduate training as a standard for all surgeons. At the Harvard School and the Massachusetts General Hospital, where I had most experience, there are 140 general ward surgical beds, 100 for the surgical specialities (orthopaedics, urology, neuro-surgery) and 300 private surgical beds. Annually there are 7,000 general surgical and gynaecological admissions. In 1939, this hospital departed from the Halsted pyramidal programme and established the *horizontal or rectangular system* because this latter eliminated or reduced the excessive and prolonged competition which was found to be soul-destroying for so many aspirant trainee surgeons. The rectangular programme depended upon two assumptions: (*a*) That men can be selected with sufficient accuracy during their final student year or during internship to assure first-rate service in a long training period, and (*b*) that it is better to offer the educational facilities of a large teaching hospital to a group of 8 or 10 highly selected individuals than to concentrate these facilities for the benefit of only 1 or 2 a year.

The present residency training programme at the Massachusetts General Hospital was outlined in the Warren Report of 1950. It provided for the *selection of* 8 *surgical house officers a year* (note there are only 2 services at the Massachusetts General Hospital whilst there are 5 such services in the General Hospital, Johannesburg), each man, so long as his performance was satisfactory, to finish *a programme of increasing responsibility lasting* 5 *years*. Included in this 5-year period may be an 'elective' year which the individual may spend at another approved institution familiarizing himself with problems of investigation or acquiring theoretical and technical skills in the basic sciences. This additional experience would naturally be for the ultimate benefit of patients and for the increasing efficiency of the hospital services. The opportunity, as here outlined, of pursuing a 5-year course of surgical training and education is of course conditional upon satisfactory performance. But no one year of that training (which includes the compulsory internship year) is considered probationary to the next in the sense that only a certain proportion of house officers will be kept on. Conversely, no appointee is under obligation to pursue the course to its finish if it becomes apparent that his abilities lie elsewhere. A small proportion alter their course for one reason or another and thus provide a certain degree of flexibility to what otherwise might become an excessively rigid and top-heavy system. In this way an occasional appointment to one of the upper rungs of the resident ladder may fall vacant.

Method of Selection

An interesting feature of the selection of surgical interns at the Massachusetts General Hospital is the selection each year of 8 men on the basis of medical-school credentials and a stiff competitive examination given by a committee of staff surgeons. All men started their training on 1 July. Applications for these intern posts came in from nearly every prominent medical school in the US and the examination and selection committees, comprised of 20 surgeons of the surgical division, were kept very busy for 2 or 3 days in making their final selection of the 8 interns who would continue as residents for a further 4 years.

I was privileged to sit in with one screening subcommittee of 4 surgeons during the intern interviews and examinations and was most impressed by the high standard. There were 72 final-year medical-student applicants from all over the country for 7 surgical appointments. A 1-hour written examination and a 20-30 minute oral examination by the subcommittee of 4 surgeons were the chief features. The final committee, under the chairman-

ship of Prof. E. D. Churchill and consisting of 6-7 senior surgeons further considered the 3-4 most promising men from each of the 3 screening subcommittees. Potential interns were interviewed a second time before a final choice was made.

Training Programme

Table I shows a representative training programme. Each general surgical service has 70 beds, and is divided into a female

TABLE I. MASSACHUSETTS GENERAL HOSPITAL REPRESENTATIVE TRAINING PROGRAMME

First Year: Surgical Intern	
General surgical ward	3 months.
General surgical emergency ward and out-patients	3 months.
Gynaecological ward	1½ months.
Fracture service	1½ months.
Neuro-surgical ward	1½ months.
Urological wards	1½ months.
Second Year: Third Assistant Resident	
General surgical ward	2 months.
Anaesthesia	2 months.
Anaesthesia emergency ward and out-patients	4 months.
Private pavilions	4 months.
Third Year: Second Assistant Resident	
Gynaecological ward	4 months.
Neuro-surgical ward	2 months.
Urological ward	4 months.
Private pavilions—gen. surgical	2 months.
Fourth or Elective Year: Second Assistant Resident	
One year in a basic-science or surgical research Laboratory or outside affiliated hospital	6 months.
Orthopaedics	4 months.
Private pavilion—gen. surgical	2 months.
Fifth Year: First Assistant Resident	
Gynaecological	4 months.
The responsible surgeon-resident for male ward	4 months.
The responsible surgeon-resident for female ward	4 months.
Sixth Year: Chief Senior Resident	

and male section. Each service has 1 senior or chief resident, 2 assistant and 2 junior assistant residents, and 4 interns. As the system of rotation is employed there are 32 assistant residents.

On the average there are about 100-125 operations performed per month in each of the two services and excluding private cases performed in the private pavilions. There are many visiting surgeons on the staff, of whom groups of 4 also have a turn at duty with each service, changing every 4 months. These visiting surgeons act in a supervisory capacity and once a week they conduct a clinical round and preside over the staff meetings. This service meeting analyses the interesting pathology encountered during the week, and discusses mortality, morbidity and problem cases. A complete typewritten statistical record is maintained and after this has been perused it is submitted to Professor Churchill for permanent filing. A list of the operations and the surgeons who performed them is also included. In addition, once a week a 1-hour grand surgical round or clinic is held in the large teaching amphitheatre, where the most interesting cases from both services are presented to a large audience of staff, visitors, students and nurses. The residents conduct these entirely and questions, opinions and criticisms are invited from the senior staff and visitors. Mistakes are freely admitted and constructive suggestions for diagnosis or treatment are put forward.

These practices could be employed with profit in all South African medical schools and teaching hospitals. In Johannesburg we only conduct private unit rounds and somehow or other the whole surgical division never seems to meet to discuss or present problem patients or a series of interesting and instructive cases. Our schools, with their different units, are too individualistic in their teachings, and that is the reason why in some of the affiliated teaching hospitals there is great diversity of opinion in the handling of many of our common problems. The department of surgery of the Witwatersrand medical school does not present to its student or postgraduate body a particular school of thought. We are still groping about trying to establish an integrated scheme of things. Fortunately we are aware of our shortcomings and, as Prof. Edward Churchill stated, 'there are scores of medical schools in the United States that are also confronted with similar problems in their constant endeavour to improve their methods of student selection, teaching and resident training'.

To revert to the Massachusetts General Hospital: The resident or registrar staff perform 98% of the surgery, and so excellent is the system of graduated training that the senior residents are competent to perform any of the major operations. The chief senior resident has the choice of the most complicated procedures and on the average performs 4 operations a week besides assisting his more junior colleagues in many others. If a very unusual operation is necessary then, on rare occasions, a senior surgeon may be invited to demonstrate it or assist an inexperienced resident. It must be realized that this 4- or 5-year training produces competent surgeons and, that with the numerous advantages at our disposal today such as better anaesthesia, antibiotics, more efficient diagnosis, and a clearer appreciation of fluid and electrolyte problems, major surgery is made safe for the patient and we no longer only depend on scintillating manual dexterity to maintain low mortality and morbidity rates. In 'small' communities this is rather irksome to senior colleagues, who will never quite appreciate that young surgeons with less experience can also produce excellent surgical results!

Example of some of the types of operations successfully performed by chief or senior residents at the end of 4 or 5 years are as follows (approximate figures only): Hernia (30-40), radical mastectomy (10), radical neck dissection (6), colectomy, right or left (6), abdomino-perineal (6), artery graft (6), aortic aneurysm (2), diaphragmatic hiatus hernia (4), total cystectomy and ureterosigmoidostomy (2), partial pancreatectomy (2), oesophagectomy (2), pneumonectomy (4), mitral valvulotomy (6), gastrectomy (20), parotidectomy (2), porto-caval shunt (3), nephrectomy (6), prostatectomy (10), hysterectomy (10), thyroidectomy (8.)

Many other operations are performed and as the residents are usually first assistants to senior surgeons with their private patients their experience in this full-time service is very extensive. It is no exaggeration to say that well over 600 major operations have been performed by a 5th year resident.

It is worth while noting that 8-12 months of a 5-year programme are spent on the private services and, because the resident is assigned to a small team of staff surgeons, he acquires some insight into the practical problems of surgeon-patient relationships in private practice, as well as adding to his technical knowledge.

At the end of a 5-year training period in an approved hospital a young surgeon qualifies to sit the American Surgical Board examinations. The 5th or 6th senior resident year is compulsory for eligibility. The American Board maintains that the most important single factor in surgical development is the opportunity under senior guidance and supervision to grow by progressive stages to the stature of complete responsibility for the surgical care of patients. Extensive major operative experience and senior responsibility is an essential part of surgical education and training. It is most desirable that the new South African College shall maintain similar ideals and not merely pattern itself on the inadequate British system of F.R.C.S. and M.Ch. diplomas or degrees, in which the emphasis is more on profound theory than the assured competent practice of surgery.

Records. During their training periods careful personnel records are maintained and at the end of each period spent in a particular service a chief's report is added as well as a record of the operations performed. During a symposium on postgraduate education at the Massachusetts General Hospital Dr. William Sweet remarked: 'There are a number of good ways in dealing with different problems and it is good for students and residents to have a broad range of thinkers about them . . . . Stifling of young creative minds by orthodox moulds of mediocrity is to be deprecated. Orthodoxy should be a guide, not a strangler, of future developments.'

Other American Hospitals

I have dealt at length with one famous teaching hospital but there are many others with similar training programmes. Professor deBakey's Department at Baylor University, Houston, includes a 6th year of training, and assignment to the thoracic service and more responsible administrative and teaching duties. The Buffalo medical school, New York, also incorporates thoracic and paediatric surgery as separate allocations to their residents. At the University Hospital of Minnesota, Prof. Owen Wangensteen conducts a weekly 3-hour conference, where the residents demonstrate interesting pathology slides or specimens, X-rays and patients; all problems are freely aired and the meeting concludes with an address by a visiting surgeon or an interesting film.

Incidentally, there are 83 medical schools in the US but probably twice as many large teaching hospitals. Some of the private clinics, particularly the Cleveland Clinic and the Ochsner Clinic, provide first-class training facilities even though they are not

undergraduate teaching hospitals. The Mayo Clinic from a postgraduate point of view is not exceptional in its provision for adequate training sufficient to satisfy the American Board of Surgery.

SUMMARY AND CONCLUSION

In summary the chief points of value in the system as I saw it were:

1. A closer liaison between the individual members of the staff of the one hospital and with the staffs of groups of affiliated hospitals.
2. A close link between the clinical services on the one hand and the pathological, radiological, chemical pathological, physiological and anatomical departments on the other.
3. A preference for clinical demonstration of various types in lieu of routine systematic course of lectures.
4. The institution of well organized, staffed and equipped research laboratories within the hospitals.
5. The gradual development of registrars so that by the end of 5 years they were very efficient general surgeons, not only in theory but also in practice.
6. The provision for first-class medical records. Dictaphones or similar types of machines were installed in various key positions such as the operating theatres and out-patient departments. Every large ward had a permanent secretary. Reports and letters were typed out without unnecessary delay.
7. A well organized photographic department for recording clinical and pathological material was readily available; likewise a surgical artist.
8. Finally, a staff of 5 full-time registrars and 4 interns to take care of 70 patients was not considered excessive. Experienced authorities pointed out with pride that, in this manner, patients were well cared for and greater progress was made in the general management of the hospital. Intelligent patients were realizing more than ever that it is specialized group medical effort or team work rather than individual physicians or surgeons who were responsible for the extraordinary advances of modern medical science. Naturally, hospital costs increased simultaneously but the all-round benefits to society and economic welfare also improved.

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THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following contributions to the Benevolent fund during July 1958 are gratefully acknowledged:

Votive cards in memory of:

Dr. F. O. Fehrsen *by* Dr. J. van Selm, Dr. A. I. Goldberg, The Physician Superintendent and Staff, Alexandra Institution, Mr. W. Coxon, Mr. and Mrs. C. Spengler, Mr. and Mrs. C. G. Karstel, Dr. and Mrs. F. K. Mitchell, Mr. Alex Dichmont, Mr. George A. Reid, The Staff of Valkenberg Hospital, The Royal Society of Health, Mr. and Mrs. L. L. Karstel, Mrs. L. F. Small, Dr. and Mrs. Shadick Higgins, Dr. E. C. Crichton, Dr. and Mrs. E. D. Cooper, Mr. S. H. Middlemost, Mr. S. B. H. Gillett, The Medical Officer of Health and Staff, City Health Department, Cape Town, Dr. P. A. Smuts, Dr. P. W. J. Keet, Dr. F. K. te Water Naudé, Mrs. I. D. Cheetham, *deur* Dr. en Mev. J. S. du Toit.

Dr. P. J. Olivier *by* Dr. and Mrs. A. W. Sichel.

Mrs. W. A. MacDonald *by* Dr. S. Cole.

Total amount received from Votive Cards £52 8s. 7d.

Services rendered to:

Mrs. H. L. Goldblatt *by* Dr. James Miller, Drs. Bok, Ofsowitz and Botha, Dr. G. Berman and Matron and Staff, Provincial Hospital, Uitenhage.

Mrs. G. Rosendorff *by* Prof. J. N. de Villiers.

Mrs. H. Schmidt *by* Dr. P. J. Joubert.

Dienste gelewer aan:

Mev. S. van der Spuy *deur* Prof. J. N. de Villiers en Drs. Woeke, Greenfield en Davis.

Mev. J. M. Joubert *deur* Drs. Paterson en Grant-Whyte.

Total amount received for Services Rendered £37 6s. 0d.

Donations:	£	s.	d.
Drs. L. Fernley, J. de V. Meiring	1	1	0
Natal Coastal Branch, M.A.S.A.	3	4	0
Northern Districts Division, Natal Inland Branch, M.A.S.A.	10	10	0
Total amount received from Donations	£14	15	0

Grand Total £104 9s. 7d.

PASSING EVENTS : IN DIE VERBYGAAN

Dr. J. C. Gilroy, M.D., M.R.C.P., formerly of 924 Philadelphia Corner, has moved to Suite 602, Medical Arts Building, Jeppe Street, Johannesburg.

* * *

Dr. Sidney Sacks, Orthopaedic Surgeon, Johannesburg, has recently returned from a 3 months' study tour of Orthopaedic Clinics in Canada and the United States of America.

* * *

Mr. I. V. Rogoff, F.R.C.S. (Edin.), Urologist, has moved from 1006 Medical Centre, Jeppe Street, Johannesburg, to 402 Osler Chambers, Jeppe Street, Johannesburg. Telephones: Rooms 22-4828 (unchanged), residence 46-9876.

* * *

Dr. D. L. Ovedoff, M.R.C.P., Physician, has moved from 32 Jenner Chambers, Jeppe Street, Johannesburg, to 402 Osler Chambers, Jeppe Street, Johannesburg. Telephones: Rooms 23-3634, residence 43-3173.

* * *

Mr. S. Skapinker, F.R.C.S. (Edin.), formerly practising at 924 Philadelphia Corner, has moved to Suite 602, Medical Arts Building, Jeppe Street, Johannesburg. Telephones remain unchanged.

* * *

Mr. David S. Davies, D.M. (Oxon.), F.R.C.S. (Edin.), Plastic and Maxillo-facial Surgeon, 805 Medical Centre, Heerengracht, Cape Town, is shortly going overseas until December. During his absence Mr. David Davies, Jnr., F.R.C.S., will carry on the practice.

* * *

Mr. Harry Fine, F.R.C.S. (Edin.), has commenced practice as a Urologist at 435 West Walk, Smith Street, Durban. Telephones: Rooms 64063, residence 35675, emergencies 29326.

Dr. Harry Fine, F.R.C.S. (Edin.), het begin praktiseer as 'n Uroloog te West Walk 435, Smithstraat, Durban. Telefoon: Kamer 64063, woning 35675, noodoproepe 29326.

* * *

Dr. G. M. Lurie, M.B., Ch.B., M.R.C.P. (Edin.), D.C.H. (Lond.), has commenced practice as a paediatrician at 909 Dumbarton House, Cape Town. Telephones: Rooms 3-1002, residence 2-6047.

Dr. G. M. Lurie, M.B., Ch.B., M.R.C.P. (Edin.), D.C.H. (Lond.), het begin praktiseer as 'n kinderspesialis te Dumbarton House 909, Kaapstad. Telefoon: Spreekkamer 3-1002, woning 2-6047.

* * *

Dr. H. J. H. (Herman) Claassens, M.Med. (O. & G.), van Kaapstad, het die M.R.C.O.G. verwerf in die jongste eksamen. Hy vertrek eersdaags na die Vasteland waar hy 6 weke lank sal vertoef.

Dr. H. J. H. (Herman) Claassens, M.Med. (O. & G.), of Cape Town, took the M.R.C.O.G. in the recent examinations. He will be leaving shortly for the Continent where he will stay for 6 weeks.

Drs. H. Penn and A. E. Amoils, Ear, Nose and Throat Surgeons, have moved their consulting rooms to 402 Medical Arts Building, corner of Jeppe Street and Troye Street, Johannesburg.

Drs. H. Penn en A. E. Amoils, Oor-, Neus- en Keelartse, het hul spreekkamers na Medical Arts-gebou 402, hoek van Jeppestraat en Troyestraat, Johannesburg, verskuif.

* * *

Dr. Edward Norman Keen, M.B., Ch.B., M.D. (Cape Town), F.R.C.S., at present Senior Lecturer in Anatomy at the University of Cape Town, has been appointed to the Chair of Anatomy in the Faculty of Medicine, University of Natal. He will assume duty on 1 January 1959. Prof. E. N. Keen succeeds his father, Prof. J. A. Keen, who retires as Professor of Anatomy of the University of Natal at the end of the present year.

* * *

Research Forum, University of Cape Town. The next meeting of Research Forum will be held on Wednesday 20 August at 12 noon in the Large lecture theatre on A Floor, Groote Schuur Hospital, Cape Town. Speaker, Dr. C. J. Uys in collaboration with Dr. J. van der Walt, Dr. G. M. Potgieter and Dr. H. Golby; subjects: 'A comparative study of the incidence of liver siderosis in the three racial groups in the Cape Town area'. Anyone interested is invited to attend.

* * *

Dr. Maxwell M. Wintrobe, Professor of Medicine, University of Utah, USA, will address the Medical Faculty of the University of Cape Town on Saturday 16 August at 11 a.m., when his subject will be 'The Spleen, Splenomegaly and Splenectomy'. He will also address the Cape Western Branch of the Medical Association on Monday 18 August at 8.15 p.m. when his subject will be 'Principles in the Management of Anaemia'. Both meetings will be held in the Physiology Lecture Theatre, Medical School, Observatory, Cape.

* * *

A course in Clinical Psychology will be held in 1959 at Tara Hospital. The course will commence in February 1959. Lectures will take place at fortnightly intervals from 8.15 to 10.45 p.m. The fee for the course is £10 10s. 0d. A similar course has been held over the last 3 years and has proved most popular and successful. The 1959 course is being organized in response to numerous requests. For details please apply to the Secretary, Medical Graduates Association, Medical School, Johannesburg; telephone 44-7040 (mornings).

* * *

Medical Library Accession Lists. Owing to the pressure of space, the publication of accession lists for the medical libraries of the Cape Town and Witwatersrand Medical Libraries will be discontinued. Any doctor wishing to have the lists will receive them on sending his name and address to the Librarian, Medical Library, University of Cape Town, Anzio Road, Observatory, Cape, and/or the Librarian, Medical Library, University of the Witwatersrand Medical School, Hospital Street, Hospital Hill, Johannesburg.

* * *

The Cape Town Mothers' Clinic, established to advise mothers on family spacing, has issued its annual report for the year ended 31 March 1958. This voluntary organization held 12 sessions a month in 6 welfare centres of the Cape Town Municipality and 1 a month in a clinic of the Cape Divisional Council. The new cases attending during the year numbered 102 Europeans and 844 non-Europeans and the total attendances 536 European and 3,500 non-European. The expenditure during the year amounted to £1,030.

* * *

4th Acqui Terme International Prize. The Azienda Autonomia of the thermal baths of Acqui Terme (natural mud baths) offers a prize of 1,000,000 Italian lira for an original (unedited) work on the articular physiopathology, clinical study and therapy of rheumatic and arthritic diseases. The prize may be awarded undivided for one work or may be divided between two entrants. Eight typed copies should be submitted.

A prize of 500,000 Italian lira, is also offered for a treatise or monograph on rheumatism. Works published during the year preceeding the closing date of the competition are also eligible. This prize will not be divided. Eight copies should be submitted, either typed or printed.

The following statement applies to both prizes: The work may be in Italian, French, English, German or Spanish, but it is preferable to use Italian or French. A resumé should be attached in Italian or French. The work should bear a device repeated on a sealed envelope containing the device and the full name and address of the author or authors. The 8 copies must be sent to the following addresses: l'Azienda Autonomia di Cura di Acqui Terme, Piemonte—Italia. The competition will close on 31 December 1958.

The Jury will be international and will consist of 6 specialists in rheumatic and arthritic diseases of world reputation and a representative of the Azienda Autonomia.

* * *

Union of South Africa, Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 25-31 July 1958. No. 31 of 1958.

Plague, Smallpox and *Typhus Fever:* Nil.

Correction (Typhus Fever).

Cape Province: Duplication. Three Native cases reported for Herschel District in Newsletter No. 29 of 1958 should be deleted as they have already been reported in Newsletter 28 of 1958.

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	2	3	1	–	6
Cape Province ..	1	4	1	–	6
Orange Free State..	1	–	–	–	1
Natal	–	–	–	–	–
	–	–	–	–	—
Totals	4	7	2	–	13

Local Authorities		*Eur.*	*Non-Eur.*
Transvaal:			
Evaton Health Committee ..	U	1	
Groblersdal District	R		1
Peri-Urban Areas Health Board (Johannesburg)	U		1
Peri-Urban Areas Health Board (Middelburg District)	R	1	
Springs Municipality	U		1
Vereeniging District	R		1
Cape Province:			
Port Elizabeth Municipality ..	U	1	2
Uitenhage Divisional Council ..	R		1
Uitenhage Municipality	U		1
Walmer Municipality	U		1
Orange Free State:			
Bloemfontein Municipality ..	U	1	

U=Urban. R=Rural.

* * *

The VI International Congress on Tropical Medicine and Malaria will be held in Lisbon, Portugal, on 5-13 September 1958. An advance notice was published in this *Journal* on 28 December 1957 (**31**, 1331), and a Second Announcement in the form of a brochure of 46 pages has now been issued, which can be obtained from Prof. Dr. Manuel R. Pinto, Secretary General of the Congress, Institute de Medicina Tropical, Lisboa, Portugal (telegraphic address Medicotropical Lisboa).

The continent of Africa, and South Africa in particular, are well represented in the extensive programme. Sir Samuel Manuwa (Nigeria) is an Honorary Vice-President of Congress, Dr. E. H. Cluver a Vice-Chairman of Division A (Tropical Medicine) and Dr. B. de Meillon a Vice-Chairman of Division B (Malaria). Amongst the Vice-Chairmen of sections are Dr. Joseph Gillman and Dr. Joseph Togba (Liberia).

The following are rapporteurs of sections or sub-sections: From South Africa Dr. R. Elsdon-Dew (amoebiasis), Dr. B. A. Dormer (mycobacterial infections), Dr. J. H. S. Gear (enteric virus infections), Dr. I. MacDonald (nutrition), Dr. O. Nestbaum (epidemiology); and from Egypt Dr. A. Halawani (schistosomiasis).

Papers will be presented by the following from South Africa: Dr. H. A. Fairbairn (trypanosomiasis), Dr. R. Elsdon-Dew (amoebiasis), Dr. G. G. Roach (amoebiasis), Dr. A. Wilmot (amoebiasis), Dr. V. Bokkenhauser (gastro-intestinal infections), Dr. B. A. Dormer (tuberculosis), Dr. C. Kaplan (tuberculosis), Capt. K. Collender (tuberculosis), Mr. A. Jennings (leprosy), Miss D. D. Cornell (leprosy), Dr. H. I. Lurie (histoplasmosis),

Dr. J. H. S. Gear (enteric viruses), Dr. H. H. Malherbe (enteric viruses), Dr. P. Smit (respiratory infections in Natives), Dr. J. Gillman (pellagra), Dr. J. A. Munoz (pellagra), Dr. F. Walt (kwishiorkor), Dr. R. A. Alexander (blue tongue), Dr. B. de Meillon (malaria eradication).

Papers will also be presented as follows: From Kenya, Dr. R. B. Heisch (tick-bite rickettsiosis), and Dr. H. R. Binns (rinderpest). From Uganda, Dr. K. C. Willet (trypanomiasis), Dr. J. A. Kinear Brown (leprosy) and Dr. A. B. Raper (malaria and sickle cells). From Nigeria, Dr. R. Lauckner (tuberculosis) and Dr. O. Ajose (health education). From Sudan, Dr. E. T. Abdel Malek (schistosomiasis) and Dr. M. H. Sati (kala-agar). From A.O.F., Dr. P. Mornet (trypanosomiasis). From Tunisia: Dr. H. Sparrow-Germa (*Borrelia recurrentis*). From Egypt: Dr. A. Halawani (schistosomiasis), Dr. A. H. Mousa (schistosomiasis), Dr. H. Foad Nagaty (ascaris), Dr. P. Ghalioungui (amoebiasis), Dr. M. A. Gohar (gastro-intestinal infections) and Dr. F. A. Assaad (vital statistics).

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

SUSTAGEN IN ULCER THERAPY

At the recent World Congress of Gastro-enterology, Dr. Asher Winkelstein of the Mount Sinai Hospital, New York, famed for his intra-gastric drip treatment for severe ulcer cases, presented a paper on the use of Sustagen (Mead Johnson) in the nutritional treatment of peptic ulcers. He worked on chronic peptic ulcers experimentally induced by repeated nervous stimulation of acid secretion.

Sustagen, as a therapeutic food for complete nourishment, contains, in precise proportions, protein, carbohydrates, vitamins and minerals, and by neutralizing free hydrochloric acid, heals the ulcer. A virtue of the treatment is that it is economical, eliminating the necessity for expensive antispasmodic drugs, alkalis and other foods. Its therapeutic results have been excellent in a large series of ulcer patients who have been treated with this method.

While the product may be considered eminently suitable as a substitute for a normal diet where a low cholesterol content is desirable, the practitioner may wish to consider sodium intake in the treatment of patients on sodium-restricted regimens. An analysis of an 8-ounce glass of Sustagen and water, is as follows: Calories 390, protein 23·5 g., fat 3·5 g., carbohydrate 66·5 g., with vitamins and minerals. The sodium content of this mixture is 210 mg.

The product is marketed in a powdered form, in 16 oz. cans. Mixed with water, it becomes a pleasant-tasting, bland liquid for both oral and tube feedings.

Pareira, M. D., Conrad, E. J., Hicks, W. and Elman, R. (1955): Cancer, 8, 803.
Winkelstein, A. and Schweiger, E. (1956): J. Amer Med. Assoc., 160, 1,111.
Winkelstein, A. (1957): Amer. J. Gastrent., 27, 45.

REVIEWS OF BOOKS : BOEKRESENSIES

BEAUMONT'S MEDICINE

Medicine: Essentials for Practitioners and Students. 7th Edition. By G. E. Beaumont, M.A. D.M. (Oxon.), F.R.C.P., D.P.H. (Lond.). Pp. xviii+847. 70 Illustrations. 45s. net. London: J. & A. Churchill Ltd. 1958.

When Dr. Beaumont set out to prepare a text book on general medicine he was singularly successful in including the essentials and excluding extraneous matter. The result was a book which has been of immense benefit to senior students and of considerable value to practitioners in establishing diagnoses. It has kept its place in medicine for over 25 years and this new seventh edition maintains the high standard one expects of it. It has been thoroughly revised and the recent advances of the last four years have been included. One finds thus that there are 28 new articles while a new chapter (Chap. VI) on Water and Electrolytic Balance, written by Dr. P. H. Friedlander, has also been included. Many other sections of the earlier edition have been rewritten, new notes added and references made to the therapeutic use of more than 30 new preparations.

The value of Dr. Beaumont's works is so well known that this new edition will be welcomed by all who have appreciated his other writings.

T.A.

FRAZER'S OSTEOLOGY

Frazer's Anatomy of the Human Skeleton. Fifth Edition. Edited by A. S. Breathnach, M.D., M.Sc. Pp. viii + 247. Illustrations 197. Many in colour. 50s. net. London: J. &. A. Churchill Ltd. 1958.

Professor Frazer's intention when writing his text-book on the 'Anatomy of the Human Skeleton,' which first appeared in 1914, was to relegate the pure description of the dry bone to a secondary place and to present the bones as they exist in the body in their intimate relationship to surrounding structures. A great attraction of this work has always been the detailed accounts of muscular and ligamentous attachments on the bones, based on specimens specially dissected for this purpose, and it is probable that no other book presents such exact analysis of these features.

The new editor has carefully maintained the original character and text of Frazer's work, perhaps too carefully as regards the text. E.g., some out of date terminology such as process of Kerckring (p. 185) and canal of Huguier (p. 192) could have been omitted with advantage. The present 5th edition has been expanded to include a number of X-rays, altogether 8 plates, each plate with 2 or 3 skiagrams. These are well chosen and illustrate anatomical levels of the skeleton, epiphyses in young bones, joints in varying positions, etc. Dr. Breathnach emphasizes in his preface that certain sections of the skull have been almost entirely re-written and that the paranasal sinuses have been given fuller treatment. Having decided on X-ray plates as a new feature, it seems strange that the author should have neglected to present at least one skiagram to illustrate the air sinuses of the skull. Other chapters which have received new treatment are those on the pelvis and the arches of the foot. In the description of the bones of the foot there is a valuable reference to recent work on the ligamentous versus muscular control in the maintenance of the arches, and the question of their relative importance seems to have been answered. When the feet support the weight of the body in standing, the arches are maintained entirely by the osteo-ligamentous mechanism for short periods of about 30 seconds, alternating with periods of muscular contractions. This has been shown by electromyograph recordings with electrodes strapped to the back of the leg. The lists of references at the end of each chapter were not seen in previous editions, and they form a valuable addition to the book.

We welcome this new edition of a well-established anatomical text-book, and have no doubt that it will continue to appeal to students studying for higher surgical qualifications, or intending to become anatomists.

J.A.K.

YEAR BOOK OF DRUG THERAPY

The Year Book of Drug Therapy 1957–1958. Edited by Harry Beckman, M.D. Pp. 518. $7.50. Chicago: Year Book Publishers, Inc. 1958.

This book consists of summaries of the most important articles relating to drug therapy published during the past year. The articles cover a wide range of medical disorders in which drugs are used, but the selection with a few exceptions is confined to literature printed in English with the accent on American work. European work is virtually neglected.

In my opinion the most valuable form for a Year Book to take would be a series of critical reviews of the work done in the various branches of a subject, rather than a collection of abstracts such as is the case with this book. However, the papers chosen for sum-

marizing are so grouped as to provide a comprehensive survey of the work done in each field. With puckish humor the author frequently places conflicting articles alongside one another. Thus an article, lauding the superiority of the new hypotensive drugs over a control series treated with phenobarbitone, is placed next to an article proving as convincingly that phenobarbitone is at least as effective as other antipressor drugs and much less harmful. Brief but pertinent comments by the editor are to be found at the end of many of the articles. These comments considerably enhance the value of the book.

In an introductory Editorial the editor delivers a spirited attack on the 'obfuscation, specious double talk and downright deceit billowing out of some of the pharmaceutical houses'. His sentiments will be echoed by many a harassed doctor overwhelmed and numbed by the propaganda which accompanies the unceasing shower of new products rained upon them.

This book should be of value to the general practitioner and specialist anxious to keep abreast of advances in drug therapy.

B.G.S.

MEDICAL TELEOLOGY

Medical Teleology and Miscellaneous Subjects. By F. Parkes Weber, M.A., M.D., F.R.C.P., F.S.A. Pp. viii+86. 15s. net. London: H. K. Lewis & Co. Ltd. 1958.

The first portion of this book is mainly a revised edition of chapters which appeared in 'Some thoughts of a Doctor'. In the second two-thirds are notes on miscellaneous subjects.

The author gives nine examples of reactions which are purposeful in the sense indicated by the title, beginning with the ophthalmic sign of death, the fragmentation of the columns of blood in the retinal vessels. This he interprets as a purposeful reaction to obstruction, using the term in its broadest sense, in this case, interference with the circulation owing to cessation of the heart-beat. He does not discuss the interesting fact that arteries are empty of blood post mortem, and are so called because of that.

In the chapter on 'Vicious circles in disease and nature's efforts to deal with them,' he builds up the conception that certain episodic diseases are the result of periodic accumulations of toxic substances which are got rid of by reactive crises. The reviewer does not subscribe to these 'purposeful' manifestations in the group of diseases discussed, nor to the author's examples and interpretations of safety-valve actions and reactions. This is not to say that he does not believe that 'blowing-off steam' and 'getting it off your chest' or aggressive reaction in certain situations may not be beneficial.

Parkes Weber is always at his best on the rare case. In the second portion of the book, 'Miscellaneous Subjects,' we have sketchy references to unusual diseases or peculiar symptoms as well as to such diverse subjects as Greek vases, collections of coins, tombstone inscriptions and remembered happenings in his intern and registrar days—a veritable miscellany.

F.F.

INTRACARDIAC ELECTROCARDIOGRAPHY

Electrocardiographie Endo-Cavitaire. Par H. Latour et P. Puech. Pp. ix plus 294. Figs. 132. 3.200 fr. Paris: Masson et Cie. 1957.

This highly technical and very specialized work is the product of two members of the Faculty of Medicine of Montpellier in France, working in the department of cardiology which is directed by Professor G. Giraud. It deals with the electro-cardiographic findings obtained by intra-cardiac electrodes, the right side of the heart being reached by venous catheterization, the left side by retrograde arterial catheterization. Different regions of the auricles and ventricles are carefully explored both by unipolar and bipolar electrodes and the findings obtained from numerous patients, suffering from diverse cardiac conditions, constitute the kernel of the book.

The factors which bring about the varying differences in potential, as revealed by intra cardiac electrography, are carefully analysed by the authors whose diligent research carries them over a wide field of investigation. The book abounds with diagrams and reproductions of electro-cardiograms from the authors' many patients and each case is discussed with meticulous detail. Each tracing receives close attention and explanation so that the book must be a very rich fund of information for those engaged in this kind of work. It certainly is a worthy addition to the list of brilliant monographs published by Messrs. Masson et Cie of Paris. A proper appraisal of its scientific value could only be offered by someone with a highly specialized knowledge and experience of electro-cardiography. These qualifications this reviewer does not possess. But I can record the impression it conveys of a scholarly survey of the electrical changes in the normally functioning heart—and that means dealing with the auricles, ventricles, aorta, pulmonary artery and other anatomical regions in great detail—ranging over hypertrophy of the auricles and ventricles, disturbances of conduction, myocardial infarct, aberations of rhythm to a lengthy consideration of the Wolff-Parkinson syndrome.

There are 283 pages of text and 132 illustrative figures, many of which have as many as a dozen electro-cardiographic tracings. While the exhibition of such a profound knowledge of this subject is a testimonial to the authors' ability and a credit to the medical faculty of Montpellier, the uneven, uncut pages in a poorly bound, flimsy, paper cover are hardly a suitable vehicle for the presentation of such a work.

C.K.O'M.

GLAISTER'S MEDICAL JURISPRUDENCE

Medical Jurisprudence and Toxicology. Tenth Edition. By John Glaister, J.P., D.Sc., M.D., F.R.S.E., F.R.F.P.S.(Glasg.), In Collaboration with Edgar Rentoul, M.B.E., M.A., LL.B., M.B., Ch.B. Pp. xi + 720. 225 Illustrations, 72 in colour. 47s. 6d. net. Postage 1s. 7d. Abroad. Edinburgh: E & S. Livingstone Ltd. 1957.

The first edition of 'Glaister' was published as long ago as 1902, and through the years there has been a steady expansion of the work to its present unique status in the field of Forensic Medicine. The 9th edition was published in 1950; there was a revised reprint in 1953, and the present edition has been fully revised with significant additions. These additions reflect the legislative changes which have taken place in the United Kingdom during recent years and include references to several areas of advance in the science of Forensic Medicine. Of particular interest is the reference to the anti-human globulin test for human blood. There seems little doubt that the use of this test in the examination of forensic exhibits may substantially improve the test criteria which have been used in the past to establish blood stains as human blood stains.

It is extremely difficult in a work of such a comprehensive nature to direct attention to specific sections of the book, but of particular value are the cases described and illustrated in the sections on deaths from drowning, suffocation, hanging, strangulation, electrical injuries, burning, fire-arm wounds and visceral injuries.

The book contains some 225 illustrations of which 72 are in colour. Many of these illustrations, and in particular those showing the characteristics of the different kinds of wounds caused by blunt force, are of an outstanding nature and should serve as a most valuable guide to students and medical practitioners.

A great deal of information has been included in the section on Toxicology, but in the very nature of this subject, with the development of new pharmaceutical, commercial and household products, it is virtually impossible to provide information which can be applied in all the emergencies of forensic medical practice. The descriptions of acute arsenical poisoning, acute carbon monoxide poisoning and acute alcoholic intoxication will be found to be of value to practitioners in the field.

Apart from the unnecessary use of 'full points' at the end of headings and sub-headings, the production of this book cannot be faulted. It is well bound and the publishers have used a most attractive double colour style on the spine. The high quality coated art paper shows the illustrations to the greatest possible advantage.

The 10th edition of 'Glaister' maintains the tradition established by the present author's father at the turn of the century, and it should prove a most useful reference work for those engaged in medico-legal practice in this country.

I.G.

SURGERY OF THE LENS

Symposium on Diseases and Surgery of the Lens. Edited by George M. Haik, M.D., F.A.C.S. and Elizabeth M. McFetridge, M.A. Pp. 260. 233 Illustrations. South African Price: £4 9s. 3d. St. Louis: The C. V. Mosby Company. 1957.

This is a beautifully produced and well-edited book. It has been evolved from a symposium on diseases and surgery of the lens

held in New Orleans. It avoids all the repetitions and over-lapping which is usually found with multiple authorship. Seven distinguished ophthalmologists, including Derrick Vail, Paul Chandler and Frederick Cordes are responsible for it.

Much space is devoted to the treatment of congenital cataract. Because of the associated abnormalities and the age of the patient, congenital cataract has a much poorer prognosis than in patients with senile cataract. Since most congenital cataracts are stationary it seems advisable to leave the patient alone if the vision is 6/15 or better. In any borderline case it is important not to hurry into operation in early life. Cordes does not operate on the first eye before 6 months of age.

Linear extraction as a primary procedure results in fewer complication than multiple needling of the lens. The necessity of a dilated pupil both at and after operation is much stressed. Atropine should not be discontinued before all the cortex is absorbed, to avoid an occlusion of the pupil. Zieglers V shaped through and through discission and intracapsular extraction are both condemned as disastrous procedures.

Techniques of cataract extraction are clearly described with much emphasis on the mechanics and reasons for each manoeuvre. This teleological approach is exceedingly helpful and rewarding to the ophthalmologist. Lowering the tension by firm pressure on the eyeball, applied after injection of the anaesthetic agent, is believed to be the most important single factor in the prevention of vitreous loss.

Perhaps the finest feature of this fine book is the round table discussion in which over 200 questions are answered.

The book must fascinate and enlighten every eye surgeon.

S.A.

SIDELIGHTS ON THE HISTORY OF MEDICINE

Sidelights on the History of Medicine. Edited by Sir Zachary Cope. Pp. x + 246. 18 Figures. 45s. + 1s. 9d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

The section of the History of Medicine in the Royal Society of Medicine has heard many papers over the past 40 years and this book presents a selection of these. Most of the papers are printed without alteration. It is quite a shock, for example, to see a reference cited as being a personal communication from Sir William Osler! The selection of papers is wide and covers many fields from Ancient Egyptian and Greek medicine to as recent events as the discovery of X-rays or the bombing of the Royal College of Physicians in the 2nd World War.

It would be invidious to select any article as more worthy of mention than any other for as might be expected a high standard is maintained throughout. But there is at least something for most of the specialties, Humphrey Davy on Anaesthesia, the parturition chair and the history of ovariotomy for the Gynaecologists, the discoverers of auscultation and percussion and the story of William Withering for the physicians, surgical lectures of 150 years ago for the surgeons—and so on. But there is much of general interest for all—the fees of our predecessors, for example!! Even the patient is not forgotten!!

It is a book for an idle moment, for a sick bed—or perhaps even to be taken in small doses as a soporific at bedtime.

M.C.

DISEASES OF CHILDREN

A Handbook on Diseases of Children—Including Dietetics and the Common Fevers. Eighth Edition. By Bruce Williamson, M.D.(Edin.), F.R.C.P.(Lond.). Pp. xi + 483. 27s. 6d. net + 1s. 6d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

This small volume, which is as remarkable as ever for its concise and nevertheless full contents, retains its familiar original form. The handy size, the arrangement of the chapters and the bold-type sub-headings are retained and greatly facilitate easy reading and quick reference. The larger size of this 8th Edition reflects new additions to the text and full revision, especially as regards therapy.

New syndromes are included and recent advances in treatment have occasioned the rewriting of many sections, bringing the book up to date. The book is well written, covering the essentials of modern pediatric practice, and is probably the best hand book on the subject of diseases of children available at present. The sections on dietetics and the common fevers are particularly noteworthy and will undoubtedly prove most useful.

This book is not intended to be a reference book for specialized graduates in paediatrics but is a concise, lucid and comprehensive manual for students, graduates and trained nurses requiring a convenient volume for study, revision or reference purposes in the field of Children's Diseases. For this purpose the book is strongly recommended.

R.M.

CORRESPONDENCE : BRIEWERUBRIEK

KATAYAMA SYNDROME

To the Editor: In a report of a case of the Katayama syndrome by Rabinowitz (1958) he stated that the purpose of the report was 'to point to the advantage of treatment at (an) early stage as a means of preventing late sequelae'. But he gives no proof of cure except to report that the patient had had no recurrence of pyrexia or ill health after one year. If a cystoscopy or rectal biopsy were performed upon his patient now, it is very likely that bilharzial tubercles would be found in the bladder or viable ova in the rectal mucosa.

In referring to my report (Walt, 1954) he states that treatment was started only after finding ova in the urine or stool. This is incorrect for 3 of my cases (3, 7 and 9) were given treatment because of the clinical picture and eosinophilia *before* positive proof was obtained. Case 9, in fact, was reported in detail because he developed haematuria 3 months after early treatment and had ova in the rectum 8 months after therapy.

Rabinowitz believes that a 5-day course of Miracil D. (Nilodin) is sufficient to cure bilharzia if given in the early phase of the disease. I refer him to the review of surgical complications by Marks (1958) and suggest that he re-examine his patient every 6 months for a few years before pronouncing him cured.

Frank Walt
125/6 Trust Buildings, Gardiner Street, Durban — *Assistant Visiting Paediatrician Addington Children's Hospital, Durban*

5 August 1958

Marks, C. (1958): S. Afr. Med. J., 32, 162.
Rabinowitz, D. (1958): *Ibid.*, 32, 658.
Walt, F. (1954): *Ibid.*, 28, 89.

BLADDER NECK OBSTRUCTION

To the Editor: In the *South African Medical Journal* of 12 July, Mr. Modlin[1] makes a prodigious and valiant attempt to cover, in 5 pages, the pathology, diagnosis, management and treatment of bladder-neck obstructions in the male—including *inter alia* the diagnosis and management of biochemical imbalances, the modern treatment of acute renal failure, and details of such highly technical procedures as transurethral resection of the prostate. I feel he must be commended for his desire to convey a picture of such a large section of modern urology, all in the same article.

As a practising urologist, I find much with which to disagree. I feel, moreover, that the least the reader can demand is that some plan of campaign for cases of bladder-neck obstruction should emerge. But much appears to have been left incomplete and vague, and certain *ex cathedra* statements require modification if not absolute contradiction. What follows is an attempt to etch in stronger lines what appears to be hazy in the picture as presented.

Questions one is repeatedly asked by students and general practitioners (and which have not here been answered) are: (1) When do you catheterize? (2) Do you empty the bladder completely or not? (3) In emptying the bladder, is it necessary to decompress slowly or not? The answers to these questions are easy, provided definitions are agreed upon and the purpose of the catheterization is kept in mind.

The purpose of catheterization of the obstructed bladder is to relieve the pain; the severer the pain, the more urgent the need for catheterization. When a painful urinary retention has been

established, history taking and all further examinations are best left until a catheter lies indwelling and urine is running merrily into some container.

Definitions

(A) Acute retention implies complete inability to pass urine with a distressed, clamant patient who presents with a full, painful, easily palpable bladder. This condition, in its most acute form, is uncommonly due to a prostatic enlargement or fibrosis but more commonly due to acute prostatitis or stone impaction; but whatever its cause, its treatment is the same, viz. immediate and complete emptying of the bladder and removal of the catheter, with urinary investigation to follow later. One attack of acute retention *per se* is certainly no indication for prostatectomy.

(B) Acute on chronic retention is the usual clinical picture, typically presenting with a story of increasing difficulty and nocturia over a period of time, culminating in complete or almost complete obstruction—perhaps a few driblets or drops of urine are passed which do not relieve the constant desire to micturate. Treatment is demanded here too, though not quite so urgently as in the first group, because the pain is not normally so intense. The treatment here is immediate and complete emptying of the bladder, the catheter being left indwelling while the patient is removed to hospital for complete urological investigation. Repeated catheterizations, with repeated risk of introduction of infection, ascending pyelonephritis and uraemia, are to be avoided in all cases of bladder-neck obstruction; it has been shown conclusively that the degree of bladder infection varies directly with the number of times the patient has been catheterized before admission to hospital.

(C) Chronic retention implies a bladder which contains a high degree of residual urine, with the patient usually quite unaware of his retention. There is nothing acute about this process, and there is never pain of the distended bladder, which is usually atonic and often quite impalpable. At times the bladder may reach as a painless swelling to the xiphisternum with minimal urinary symptoms. There is, according to our conception of the indication for catheterization, no reason whatsoever to catheterize this patient; in fact, any interference in this type of case is fraught with the gravest risks, and any catheterization outside a hospital, and before a complete urological investigation has been undertaken, is, to say the least, meddlesome in the extreme.

Immediate suprapubic cystotomies are usually eschewed today, the difficulty of catheterization having been overcome, in the complicated case, by the use of a small Tiemann catheter. All that can be said of a suprapubic puncture, is that, dramatic as it may sound, it has never been required in the experience of the majority of urologists.

The bogey of haematuria and acute renal failure following catheterization and immediate complete emptying of the acute, or acute on chronic, retention has been well and truly laid, particularly as the result of the work of Wilson Hey and Charles Wells; any haematuria occurring after the introduction of a catheter is prostatic in origin and not from the kidneys, and the haematuria occurring later may in addition be due to an ascending pyelonephritis following infection. The haematuria is not due to rapid emptying of a dilated upper urinary tract. Quite apart from this fact, however, it has been realized for a long time that gradual decompression of the bladder, as a practical procedure, is quite impossible technically either by catheter drainage or suprapubic cystostomy. When Dr. Modlin states that the indwelling catheter carries a high risk of infection in these patients and is undesirable—referring I assume to patients in acute renal failure who are also obstructed at the bladder-neck—he omits to suggest the form of drainage these people are to have while he carries out his multiple procedures for restoration of renal function.

While 'haematuria may occur from an enlarged prostate and is painless and bright red in colour' it cannot be overstressed that papillomata and carcinoma of the bladder are far commoner causes; the enlarged prostate is an extremely rare cause of painless haematuria and should be considered such.

'Perurethral resection followed by hormone therapy is the correct form of treatment for carcinoma of the prostate.' This statement is quite unacceptable and, according to figures from Boston and the Walter Reed Hospital, both untrue and misleading. Radical prostatectomy is the treatment of choice for operable carcinoma of the prostate; this operation produces 50% cures at the end of 5 years as opposed to the 26% living (but not cured) where palliative measures have been tried. In the inoperable cases it has been shown conclusively that treatment by orchidectomy and oestrogens, with or without transurethral resection, provides a longer survival than by oestrogens and transurethral resection only.

Finally a word about that very important procedure of catheterization and its technique. As trauma should be avoided at all costs, the catheter used should be one designed to overcome prostatic obstruction. This is the small Tiemann catheter, either in rubber or plastic, which will negotiate almost all adenomatous glands, provided it is passed with the point up, from the bulb onwards. It will certainly pass where no blunt-tipped Foley catheter can go and should be the catheter of choice. Absolute asepsis by all means—but certainly neither masks, caps nor sterile trollies are available in farm-houses or private homes, where the majority of catheterizations are of necessity carried out. Let us therefore still maintain some slight sense of proportion in the matter.

I. Jacobson

706 Medical Centre
Cape Town

1 August 1958

1. Modlin, M. (1958): S. Afr. Med. J., 32, 702.

URINE EXAMINATION FOR INSURANCE

To the Editor: May I request the Chief Medical Officers of Insurance Companies to issue standing orders to their offices and agents to warn their clients, before bringing them for examination, that the urine *must* be examined before they can be passed. In many instances the candidates are unable to pass urine at the time of examination for the reason that they were not told about it. This constitutes a nuisance to the medical examiner, and also to the candidate, who finds it necessary to come back

Medical Examiner

4 August 1958

MEDICAL INSURANCE SOCIETIES

To the Editor: My attention has been drawn to notices which have appeared in the issues of your *Journal* dated 21 June[1] and 12 July[2] 1958 referring to the practice of Medical Insurance Societies in regard to the application of the Medical Aid preferential tariff.

It is alleged that the companies are endeavouring to undermine the basis on which Medical Aid practice is recognized by the Medical Association of South Africa. It is also suggested that medical practitioners who agree to accept payment in accordance with the preferential tariff applicable to approved Medical Aid Societies, will lay themselves open to being coerced to accept even lower fees at some future date.

I would like to assure you that my Company has no intention of exerting any pressure whatsoever and we are concerned that we should be accused of undermining medical aid practice. The practice of my Company in regard to the payment of the claim moneys was adopted after numerous requests of medical practitioners had been received, who expressed the wish that the proceeds of the claim should be paid directly to them and not to their patient. It was certainly not our intention to try and undermine the basis on which Medical Aid practice is recognized. In the circumstances my Directors have now decided to discontinue the notice and authorization form.

It is possible that certain of the special forms are in the possession of policy holders who are undergoing treatment at the present moment. We should accordingly like to ask you to announce through the medium of your *Journal* that our practice has now been changed and to ask medical practitioners on our behalf to ignore the red printed slip attached to a claim form if a patient should affix it to his claim form.

Dr. G. H. Hansmann
Managing Director

S.A. National Sickness and Accident Insurance Co. Ltd.
P.O. Box 1, Sanlamhof, C.P.

1 August 1958

1. Medical Insurance Societies (1958): 32, 631.
2. Association News (1958): 32, 712.